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EMPYEMA AND THE UNEXPANDED LUNG FOLLOWING ARTIFICIAL PNEUMOTHORAX FOR TUBERCULOSIS

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THE title of this study should be qualified to read "The problem of tuberculous empyema and the unexpanded lung following artificial pneumothorax in the experience of a large municipal hospital." Some consideration must be given to the magnitude and character of admissions to such a hospital treating tuberculosis. During the past five years 3,000 patients have been admitted each year to the tuberculosis wards of Bellevue Hospital where this study was carried out, and during this same period artificial pneumothorax was initiated on approximately 350 patients a year. In addition, on the wards, as well as in the pneumothorax clinic attached to the hospital, some 500 patients each year received refills of air in the course of treatment.

As to the character of these admissions it must be stressed that a very small proportion of these patients began, continued, and completed their pneumothorax period at Bellevue Hospital. In a sense the hospital acts as a clearing station where a large number of patients are received from other hospitals, or transferred to various institutions in the course of treatment, but only a relatively small number are carried completely through this period. Also, because of the shifting nature of the population from which these people are drawn in New York City and because of the policy of redistricting the city for the care of tuberculosis, almost one-half of the patients treated in the outpatient pneumothorax clinic of the hospital have shifted elsewhere in the course of treatment. Therefore, any study contemplated at such an institution necessarily must be focused on various phases during the

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course of treatment in these patients. As a result, the statistics as given in this study are not relative to the number of patients receiving artificial pneumothorax but are pertinent only to each phase as discussed. Furthermore, the criterion for considering a case as empyema was simply the presence of thick, purulent pleural fluid for which treatment was deemed necessary. Fluids, even though turbid and containing tubercle bacilli, were not considered as empyemas. Mixed tuberculous empyemas were designated as such because of the presence of pyogenic organisms on culture of the purulent pleural fluid. The term "unexpanded lung" is used to describe the permanently collapsed lung with a free pleural space containing fluid.

During the past five years 137 patients with tuberculous and mixed tuberculous empyemas were admitted to the wards at Bellevue Hospital. This represents 0.91 per cent of the 15,000 patients admitted with a diagnosis of tuberculosis. The incidence of empyema on the basis of initial pneumothoraces given was 7.8 per cent, or 137 of 1,750. This figure is inaccurate because of the lack of follow-up throughout the course of pneumothorax for the reasons just stated. The incidence of empyema as reported by several authors varies from 5 per cent to 14 per cent (Table I).

TABLE I

AUTHORS	INCIDENCE OF EMPYEMA (PER CENT)
Leaver and Hardaway	5.8
Mainin	5.8
Peters	10.4
Ferrari	9.0
Burrell	10.8
Brock, Muellen, Woodson	14.4

TABLE II

TYPES OF EMPYEMA FOLLOWING ARTIFICIAL AND SPONTANEOUS PNEUMOTHORAX

	TUBERCULOUS EMPYEMA	MIXED TUBERCULOUS EMPYEMA	TOTAL
Following artificial pneumothorax	72	71	143
Following spontaneous pneumothorax	6	18	24

To this group thirty additional patients who had been treated surgically during the five previous years have been added to make 167 cases as a basis of a part of this study. In twenty-four of these patients empyema did not follow artificial pneumothorax but was due to spontaneous pneumothorax. In other words, 143 cases, or 85.7 per cent, of all the empyemas were complications of artificial pneumothorax.

As shown in Table II, approximately one-half of the 143 cases of empyema were classified as straight tuberculous empyema. Since these

patients raised problems different from those raised by patients with mixed empyema, they will be discussed separately.

In this group it was found that the greatest proportion (fifty-eight cases) of those who developed empyema followed the progression of effusion incident to artificial pneumothorax. Other special causes which have been listed are closed pneumonolysis (three cases) and neglected re-expansion (nine cases). Under the heading of neglected re-expansion are listed those patients who had had their pneumothorax discontinued but were not closely followed during the period of re-expansion. These patients entered the hospital with tuberculous empyema from one to three years after an unfollowed re-expansion. It is interesting to note that in this group there were six patients with bronchopleural fistula (proved by the dye method) who did not have mixed infection.

TABLE III

MODE OF ONSET AND TREATMENT OF TUBERCULOUS EMPYEMA

CAUSE OF ONSET	NO. OF CASES	TYPE OF TREATMENT					
		NONSURGICAL				SURGICAL	
		TAPPING	IRRIGATION	DYE	OLEOTHORAX	THORACOPLASTY	THORACOPLASTY AND THORACOTOMY
Following persistent effusion	58	24	12	1	3	8	10
Following Jacobeus	2	1		1		0	0
Following spontaneous pneumothorax	3		3			0	0
Neglected expansion	9	5		1	1	1	1
Total	72	30	15	3	4	9	11

Of the surgically treated patients in this series of twenty with tuberculous empyema, nine, or 45 per cent, were cured. Six patients, or 30 per cent died, and five were unimproved. An examination of the mortality group revealed that three died of contralateral spread; one, of contralateral spread plus amyloid disease; one, from the shock of operation; and one, of sepsis following wound infection. In eleven of this group of twenty patients, thoracotomy had to be employed in addition to thoracoplasty. Open drainage immediately brings with it pyogenic infection and its complications. This is shown by the fact that five of six deaths occurred in the eleven patients on whom drainage became necessary. In the group treated nonsurgically there is no adequate follow-up to the completion of treatment for empyema and re-expansion of the lung which is the ultimate goal of nonsurgical therapy.

Therefore, to supplement this group ninety-eight patients from the outpatient clinic were studied in the termination phase of artificial pneumothorax. These had had artificial pneumothorax from two to

eight years or more, and all with sputum negative for tubercle bacilli had reached the stage of re-expansion. They have been tabulated as expanded, expanding, or failing to re-expand (Tables IV and V).

TABLE IV

EXPANSION STATUS OF 98 PATIENTS IN TERMINATION PHASE OF PNEUMOTHORAX

	NO. OF YEARS OF PNEUMOTHORAX							TOTAL
	2	3	4	5	6	7	8 OR MORE	
Re-expanded	5	8	24	10	13	6	1	72
Re-expanding	3	0	2	2	0	0	0	7
Failing to re-expand	0	0	0	4	4	4	7	19

TABLE V

RELATIONSHIP OF PREVIOUS EMPYEMA TO RE-EXPANSION

	NO.	EMPYEMA IN COURSE		TREATMENT FOR EMPYEMA
		NO.	PER CENT	
Re-expanded	72	3	4.2	2 tapped; 1 irrigated
Re-expanding	7	2	28.5	1 tapped; 1 irrigated
Failing to re-expand	19	8	42.2	Tapping in all
Total	98	13	13.3	

In this group nineteen failed to re-expand and were left with an unexpanded lung with a free pleural space. A further analysis showed that thirteen patients had had tuberculous empyemas during the course of pneumothorax. In the expanded group only 4.2 per cent had had empyema, whereas in the group failing to re-expand 42.2 per cent had had tuberculous empyema. These patients had simple and only slightly toxic tuberculous empyema and were treated by tapping or irrigation. In this most favorable group of tuberculous empyemas the relationship to the nonexpansion of the lung is shown.

There was another group which received nonsurgical treatment, and all of these patients have been followed. This group included twenty patients who received oleothorax for treatment of tuberculous empyema.

TABLE VI

RÉSUMÉ OF 20 PATIENTS WITH TUBERCULOUS EMPYEMA TREATED WITH OLEOTHORAX

	NO.	BRONCHIAL FISTULA	OTHER COMPLICATIONS		
			FIBROTHORAX	SPREAD	FLUID
Re-expanded	3		2		
Failing to re-expand	11	1		1	5
Thoracoplasty	4	1			
Unknown	1			1	
Died	1		2	1	
Total	20	2		3	5

In this group only three of those treated re-expanded to obliterate the pleural cavity, while eleven failed to expand and four were operated upon and collapsed by thoracoplasty. It may be added that, of these eleven patients with lungs failing to re-expand, six have been

advised to have a thoracoplasty for collapse of the free pleural space and lung. Only one died, and two developed bronchial fistulas.

It is evident that the majority of tuberculous empyemas follow a persistent pleural effusion. As has been pointed out by various observers, the incidence of pleural effusion is high following artificial pneumothorax and varies between 40 and 100 per cent. The persistence of an effusion, however, should be a warning, and it is at this time that decision should be made as to the course of treatment to be employed to avoid tuberculous empyema. If collapse of the underlying lung is inadequate and, as is most often true, an apical cavity is being held open by adhesions, the pneumothorax should be abandoned in favor of local thoracoplasty when possible. Furthermore, it should be determined at that time whether the lesion of the underlying lung, although well collapsed, would ever permit of re-expansion. If not, some other means of collapse should be considered.

With the development of the tuberculous empyema those patients should be selected for nonsurgical treatment who have a good collapse of the pulmonary lesion and whose lesion would permit eventual re-expansion. Even in these cases there is a probability that the end result will be an unexpanded lung with free pleural space. What actual hazard there may be in allowing a patient to continue with an unexpanded lung could not be determined from the material available, nor could any evidence be found in the literature on this subject.

TABLE VII

MODE OF ONSET AND INCIDENCE OF BRONCHIAL FISTULA

MODE OF ONSET	NO. OF CASES	BRONCHIAL FISTULA PROVED BY DYE METHOD
No definite cause	30	16
Following treatment for tuberculous empyema	20	7
Episode of spontaneous pneumothorax	10	6
Jacobeus operation	1	0
Refills of air	6	4
Unexpanded lung	4	3
Total	71	36

In the group of seventy-one cases of mixed tuberculous empyema, however, there were four instances in which the mode of onset was the development of a mixed tuberculous empyema in the presence of an unexpanded lung. All four of these cases had negative sputa for periods of two, three, and five years' duration and had shown no expansion of the lung from six months to three years before the onset of the mixed tuberculous empyema. In addition, there were twenty patients who developed pyogenic infection during the course of treatment for tuberculous empyema. The other causes of onset are listed as internal pneumonolysis, refills of air, and definite episodes of spontaneous pneumothorax.

It is interesting that, in the six patients who developed an acute empyema twelve to forty-eight hours following a refill, a bronchial fistula was proved in four. Contamination at the time of refill seems less likely as a causative factor than does the occurrence of spontaneous pneumothorax, possibly due to tearing of adherent lung at the time of refill or shortly thereafter. The incidence of bronchial fistula proved by the dye method was 50.7 per cent.

Fourteen patients in this group did not receive surgical treatment; four died in the hospital, and, of the ten remaining, all of whom were transferred to other institutions, two were improved in that the empyema had been rendered sterile. The others were unimproved at the time of transfer and probably died.

Fifty-seven patients with mixed tuberculous empyema were treated surgically.

TABLE VIII

RESULTS OF SURGICAL TREATMENT OF MIXED EMPYEMA

	NO. OF CASES	CURED	IM, PROVED	UNIM-PROVED	BRON-CHIAL FISTULA	DIED	MOR-TALITY (%)
Intercostal drainage	3	0	0	0	1	3	79
Thoracotomy	21	0	0	8	13	13	
Thoracoplasty without drainage	1	1			0		
Thoracoplasty with drainage	18	4	1	4	8	9	
Schede operation after thoracoplasty	14	5	6	1	10	2	33
Total	57	10	7	13	32	27	

TABLE IX

MORTALITY OF SURGICALLY TREATED MIXED EMPYEMA

CAUSE OF DEATH	NO. OF CASES	AUTOPSY
Postoperative bronchopneumonia	1	1
Spread of tuberculosis contralateral post-operatively	2	0
Shock of operation	2	0
Hemorrhages from wound	1	0
Continued sepsis	4	1
Amyloidosis	5	5
Meningitis	1	1
Staphyloccic septicemia	1	1
Progression of pulmonary disease	7	3
Unknown	3	0
Total	27	12

In this group 47.3 per cent of the patients died, and 17.5 per cent were cured. Another 12 per cent have negative sputum and are in the course of completion of operation for residual sinuses; this represents the improved group. It is interesting to note that in the ten cured cases the average time from onset of empyema to cure was three years and the average number of operations was between four and

five. It is more than likely that those listed as unimproved will die of the disease. The highest mortality, of course, existed in those who had only drainage; when that group whose mortality was seventy-nine per cent is excluded, we find that, of the others whose condition permitted thoracoplasty, only 33 per cent died.

On examination of the causes of death of the twenty-seven in this group of patients treated surgically, six can be said to have died directly from the effects of the operative procedure, while the remaining eighteen died of sepsis or progressive tuberculosis.

In this most desperate type of empyema following artificial pneumothorax the treatment differs according to the presence or absence of bronchial fistula. In those cases without bronchial fistula, the employment of tapping and irrigation with gentian violet and other chemicals should be utilized. This method not only renders the pleural space sterile in many instances but also has the advantage of demonstrating, through expectoration of the dye, the presence of a bronchopleural fistula. This method, however, should not be employed for more than a week when the sepsis is not controlled. Prolonged treatment by this or any other method of aspiration and irrigation is to be deplored in the face of continued sepsis and loss of resistance in a patient whose chance for control of the underlying pulmonary lesion may be jeopardized. In those patients controlled quickly by this means and the pleural fluid rendered sterile, the problem then reverts to that of the treatment of a tuberculous empyema. Here again it is believed that in those cases where collapse is not entirely effective or subsequent re-expansion of the pulmonary lesion is deemed inadvisable, thoracoplasty should be performed without delay. Even when a good collapse is present, and the lesion should permit re-expansion in the course of time, we feel that this is the most favorable time to obliterate the pleural cavity and maintain collapse of the lung. The alternative course rests in the chance of effective nonsurgical treatment of the tuberculous empyema with the possibility of development of a full-blown bronchopleural fistula later, even though not demonstrated at first. At the extreme end of the picture, furthermore, is the probability of an unexpanded lung as a final result.

In those cases not responding to treatment by irrigation and gentian violet or other chemicals, prompt and adequate drainage is of first importance. This is usually accomplished best by means of open thoracotomy at the most dependent point of the empyema cavity. Intercostal drainage has been employed for varying periods of time in twelve of the cases studied but in every instance was followed by thoracotomy for more adequate drainage. It is true that the intercostal closed drainage was of siphonage type and that suction and routine tidal drainage methods were not used. Control of sepsis, however, in these patients is so important that any procedure short of

thoracotomy seems in most cases a loss of valuable time, except as a preliminary procedure when it is thought that open thoracotomy would not be tolerated.

Of next importance to drainage is the reduction of the secreting surface of the infected pleura by means of graded thoracoplasty as soon as the condition permits. Absence of early collapse not only permits continued toxicity from infected surfaces but allows actual loss of protein when the drainage is profuse. Two patients have been seen recently who developed marked edema with reduction in the total protein of the blood serum and reversed serum albumin-globulin ratio. In both of these patients the Congo red test showed no evidence of amyloid disease.

The collapse of the pleural cavity is best accomplished by clean procedures above the area of drainage. The use of an anterior thoracoplasty through the axillary approach with removal of the anterior segments of the first four ribs is the procedure of choice for the first stage because it is associated with relatively little postoperative reaction and permits an easy and early second stage posteriorly with removal of the transverse processes and remainder of ribs. In addition to graded thoracoplasty these cases usually require a plastic or some modification of a Schede thoracoplasty to complete the closure of the pleura and healing of all the sinuses. The presence of a definite bronchopleural fistula which occurred in 50 per cent of this series of mixed tuberculous empyema is an indication for prompt surgical drainage to be followed by thoracoplasty as outlined above. In addition to the risks of sepsis and lowered resistance to the progression of the pulmonary disease, there is the ever present danger of bronchogenic spread in, if not flooding of, the pulmonary bed. The Schede thoracoplasty with muscle transplants for closure of the residual bronchial fistula is responsible for the ultimate cure of most of these patients.

SUMMARY

1. There is an inadequate follow-up of cases of pulmonary tuberculosis treated with artificial pneumothorax in a large municipal hospital. There is no continuity of treatment in one institution because of the necessity of transference of patients to other hospitals during the course of the pneumothorax treatments and because of the shifting and redistricting of the clinic population.

2. During the past five years, 137 patients with tuberculosis and mixed tuberculous empyema represent 0.91 per cent of 15,000 admissions with tuberculosis or 7.8 per cent of the 1,750 patients given initial pneumothorax during this period at Bellevue Hospital.

3. Eighty-five and seven-tenths per cent of the 167 empyemas studied complicated the management of artificial pneumothorax, and they were

equally divided into tuberculous empyema and mixed tuberculous empyema groups.

4. In tuberculous empyemas the most common mode of onset was found to be a progression of persistent pleural effusions during the course of artificial pneumothorax. The surgical results in this group have been reviewed.

5. Ninety-eight cases in the termination phase of artificial pneumothorax and twenty cases treated for tuberculous empyema by oleothorax have been presented. The relationship of an unexpanded lung and tuberculous empyema has been demonstrated.

6. A statistical study of the dangers dependent upon the unexpanded lung with free pleural space could not be carried out with the material available nor could any evidence be found in the literature.

7. In four instances the unexpanded lung was found to have preceded mixed tuberculous empyemas in the seventy-one cases of this type of empyema.

8. The results of the surgically treated group of mixed tuberculous empyemas have been analyzed and the methods of treatment discussed in the absence or presence of bronchopleural fistulas which were demonstrated in 50 per cent of these patients.

CONCLUSIONS

1. Artificial pneumothorax should be instituted in municipal clinics only where there is an associated hospital, and there should be continuous follow-up to termination of care.

2. The presence of a persistent effusion during the course of artificial pneumothorax should be considered a warning, and careful consideration of subsequent treatment should be made.

3. The unexpanded lung is a complication of tuberculous empyema, and more evidence must be obtained to determine the actual danger of this state.

4. The advent of tuberculous empyema during the course of artificial pneumothorax is a serious complication. It is considered that the best opportunity for cure rests in collapse of the pleural space and underlying lung by thoracoplasty at this time rather than in the continued artificial pneumothorax with the risks of progression of tuberculosis and development of a bronchopleural fistula.

5. Mixed tuberculous empyema without bronchopleural fistula must not be treated by nonsurgical methods unless the pyogenic infection and sepsis is controlled within a week by this means.

6. Mixed tuberculous empyema with bronchopleural fistula must be treated immediately by surgical drainage to be followed by thoracoplasty.

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DISCUSSION

DR. B. NOLAND CARTER, Cincinnati, Ohio.—My remarks will deal with some of the aspects of the surgical treatment of the empyemas which have been discussed. First, however, I should like to show a slide on which have been placed the results of a study made at the Hamilton County Tuberculosis Sanatorium on the incidence of empyema in patients who are receiving artificial pneumothorax. (Slide) In 898 cases treated by artificial pneumothorax, ninety-four, or 10 per cent, have developed empyema, and 20 per cent of these with empyema had lesions which were classified as fibroid, whereas 80 per cent had caseous bronchopneumonia either alone or associated with fibroid lesions. One-half of the patients with empyema did not have cavities in their lungs. The two facts which these studies have shown are (1) empyema has followed pneumothorax in a much higher per cent of cases than we thought and (2) empyema develops much more frequently when the lesions in the lung are in the acute stage.

It has been our feeling that the pure tuberculous empyemas do unusually well after an extrapleural thoracoplasty, and it has been our custom to perform such operations as soon as we find that other simpler methods are not going to be effective. In general, in such an operation the transverse processes are not resected, and the segments of ribs removed are not as long as those which are taken in the usual thoracoplasty. Segments of ribs 1 to 10 are removed in three stages as a rule. (Slide) This slide brings out rather strikingly the results of the various types of treatment for the pure tuberculous empyema. The indifferent treatment had a mortality of 97 per cent with no recoveries; the oleothorax, 35 per cent mortality with 32.5 per cent recovery, and the thoracoplasty, 10.7 per cent mortality with 68 per cent recovery.

The most difficult group of cases to handle are the mixed infection empyemas. The patients are poor risks; the empyema cavities are huge; the pleura is very thick; and the lungs completely fixed. An important point to bear in mind is that, although these patients may respond quickly to upbuilding measures, such as adequate drainage, rest, and blood transfusion, they even then will tolerate relatively little in the way of operative procedures. For this reason their operations should be broken up into many easy stages rather than into a few dangerously risky ones.

In our hands extrapleural thoracoplasty alone has not been a satisfactory operation for this type of empyema except in the very early cases. Although we have

been able to decrease the size of the empyma cavity by its use, we have not been able to collapse completely the walls of the cavity, the result being an empyema cavity of the same general shape as the original one but of a smaller size. In our opinion it is the tremendously thickened pleura which prevents the complete collapse of the cavity, even though the bony roof over the cavity has been removed.

For all mixed empyemas, save the very early ones in which there has not been time for great thickening of the pleura to take place, we have been using the many staged operation in which both the ribs and the thickened pleura overlying the empyema cavity are removed and the cavity filled with muscle and skin flaps. At the first stage adequate drainage is procured by resecting long segments of two ribs and the underlying pleura. The cavity is rendered as nearly sterile as possible by active treatment with Dakin's solution or azochloramide over a few weeks' time, when the subsequent stages are carried out as rapidly as the patient can tolerate them.

(Slide) This slide shows the results we have had with this type of operation. In Group 1, with very little active lung involvement, the results are naturally good as to recovery, because we feel we can always obliterate the empyema cavity, although it may take many operations to do so. In Group 2, seven of the ten patients died, as a rule, of some complication of the diseases, such as meningitis, spread, or peritonitis, before the series of operations could be completed.

DR. LOUIS R. DAVIDSON, New York City.—Dr. Auerbach, in a recent paper describing 1,000 consecutive autopsies at Sea View Hospital, indicated that 7.8 per cent of all those cases had bronchopleural fistulae. I should like to confine my remarks solely and exclusively to this question of mixed infectious tuberculous empyema.

One point that must be considered is whether or not the patient has positive sputum. We have found at Sea View Hospital, in a series of 125 cases of mixed infectious tuberculous empyema, that 54 per cent of our patients had positive sputum and that 46 per cent had negative sputum.

Now, the question is, What shall we do with these cases? Sixty-seven per cent of these patients with mixed infectious tuberculous empyema had bronchopleural fistulas by examination—that is to say, these patients expectorated methylene blue. It is our belief that *all* empyemas which follow pneumothorax therapy are the result of bronchopleural fistulas whether or not these are clinically proved. In fact, it is our operative experience to find bronchopleural fistulas in cases in which clinically it was *impossible* to prove their existence. We have come to the philosophy that in these cases of mixed infectious tuberculous empyemas there is only one treatment, because we feel that almost all of these cases are based upon the present or prior existence of a bronchopleural fistula. The pyogenic infection is not introduced from outside. In order to prevent the ever present possibility of contralateral bronchogenic spread we operate immediately. First we perform an anterior thoracostomy and about four days later we do a thoracoplasty. We do *not* stop at three ribs. We remove six, seven, eight, or even nine ribs if the empyema is a universal affair. Then, ten days after that, if possible, we perform a saucerization Schede operation.

The reason for the urgency of this particular type of procedure is that the periosteum which has remained has had no time to regenerate. With the scalpel one can definitely, easily, and quickly unroof the empyema space. Then the space is completely packed.

However, some of these cases come to what we term the plastic operation. We have abandoned the primary Schede operation with muscle flap in the early stages. We have had a very definite number of cures. The figures are something like this: in 125 cases, thirty-nine of which had only a thoracostomy, thirty-nine or 100 per

cent died. We feel that it is futile and serious to establish merely drainage. For example, we do not perform a thoracoplasty in a patient with a badly involved bilateral caseous pneumonic tuberculosis because the patient usually succumbs to the disease, surgery, or both. We also feel that it is as futile to attempt to do any surgery in a patient with marked empyema on one side and a large cavity on the other side. Furthermore, thoracostomy is painful, and the dressings are frequent. The patient is markedly uncomfortable. We should much prefer to permit the patient to die rather than to torment him further with unavailing surgery.

Eighty-six cases remained for surgery. The Schede operations were divided into three stages. In the first stage, or early days, we closed the space entirely. In the second stage of development the pleural space was permitted to remain partially open. In the third stage of treatment the space was permitted to remain open (saucerization Schede).

Of these eighty-six patients, fifty-six eventually came to Schede operation; of these, ten patients died, thirty are cured, fifteen are still in the hospital, and one is A. O. R. If we assume that the fifteen patients in the hospital at the present time and the one A. O. R. will go on to cure, as we believe they will, the statistics will then read as follows: in these cases of mixed infectious tuberculous empyema coming to Schede operations, the possibility of cure will be 82 per cent; in those cases going *beyond* the thoracostomy stage, the cures will be 54 per cent.

I venture to say that in the near future, in the treatment of mixed infectious tuberculous empyema, the great majority of surgeons will be doing an open saucerization Schede operation. Some cases will need a final plastic operation. I sincerely believe that the Schede operation is the only solution for this very serious mixed infectious tuberculous empyema at the present time.

DR. DEAN B. COLE, Richmond, Va.—I wish to congratulate the authors for the presentation of this timely paper.

For the past fifteen years we have treated tuberculous empyema on the theory of prevention and in private practice have reduced the incidence from an original 8 per cent to a temporary 0. We realize that the occurrence of an occasional tuberculous empyema is inevitable, but we are convinced that the percentage can be kept exceedingly low by keeping the pleural cavity dry. A clear transient fluid, small in amount, requires no special consideration, but any persistent fluid requires aspiration. All turbid or cloudy fluids require frequent aspiration and, if purulent, irrigation. This should be done daily and sometimes more often until fluid clears or disappears. Most bronchopleural fistulas are small in the beginning and will enlarge if neglected, but they will close if properly and adequately treated. The large tear from spontaneous pneumothorax is the exception, and, when this occurs, more radical treatment may be required.

Persistent tuberculous infection of the walls of an empyema may resist treatment, and also, because of excessive scar tissue and fistulous tracts communicating with neighboring structures such as infected ribs, cartilage, sternum, spine, or suppurating mediastinal lymph glands, would continue to feed the empyema unless and until the focus is removed. These complications fortunately are rare, but, when encountered, require immediate surgical treatment.

In summary, 90 per cent of all fluids complicating therapeutic pneumothorax show tubercle bacilli on culture, and, because of the almost constant presence of tubercle bacilli in pleural fluid complicating therapeutic pneumothorax in tuberculous patients, the continued presence of fluid is always a hazard and a potential menace in the development of empyema. Also, most pleural adhesions complicating pneumothorax and preventing lung re-expansion are the result of fluid.

Ineffectual pneumothorax should be abandoned or supplemented.

DR. JEROME R. HEAD, Chicago.—I believe that I am to speak later this morning. For this reason I hesitate to take additional time now. I do so only because I disagree so directly with some of the statements which have been made in this discussion.

I believe that tuberculous and mixed infection empyema, without bronchial fistula, occurring as complications of artificial pneumothorax, should be treated by daily aspirations and irrigations. I am convinced that drainage and thoracoplasty are rarely indicated and that neither should be resorted to until the simpler measures have been given adequate trial.

Mixed infection empyema with bronchial fistula presents a different problem. These require some variety of valvular drainage, and not infrequently the chronic empyema which persists must be treated by thoracoplasty. But to consider even this condition as an indication for immediate radical resection of the chest wall, in my opinion, is ill advised and dangerous. On simple drainage many of these patients will become afebrile and will put on weight. Over a period of months the cavity will decrease in size. Operation can then be performed at a chosen time when the patient is in excellent condition and the chances for success are greatly improved.

The only good reason for losing any of these patients is the existence of such extensive tuberculosis in the opposite lung that this original disease determines the result.

During the past eight years at Edward Sanatorium we have had twenty cases of mixed infection empyema complicating artificial pneumothorax. Many of these had the complication at the time of their admission to the Sanatorium. Four had demonstrable bronchial fistulas, and sixteen had none.

Of the first group, two were treated by drainage and subsequent thoracoplasty. Both recovered. Two had such extensive tuberculosis in the opposite lung that only palliative treatment could be considered. These died of tuberculosis.

All of the sixteen patients without bronchial fistulas recovered. Formerly such patients were treated by drainage and thoracoplasty. During the last five years the results from irrigations with azochloramide have been so satisfactory that we have had to resort to the more radical treatment in only one instance. This was the only case infected with the pneumococcus.

Our technique is as follows: With the patient in the upright position, the aspirating needle is inserted at the base of the empyema cavity and all of the pus is aspirated. Through the same needle several hundred cubic centimeters of azochloramide are injected, and this fluid is reaspirated. This procedure is continued until the fluid returns clear. About 100 c.c. of azochloramide are left in the pleural cavity. About twice a week at the final instillation 1:5,000 gentian violet solution is used. In resistant cases a small amount of cod-liver oil is injected at the end of each aspiration. Under this treatment, in most instances, the fever returns rapidly to normal, and the pleural cavity becomes sterile in the course of a week or ten days.

DR. JOHN N. HAYES, Saranac, N. Y.—I am especially interested in the unexpandable lung, with an effective collapse in a large pneumothorax pocket. I have found that it could hardly be expected that thoracoplasty would be able to bring the chest wall down onto the lung, and I have seen some patients with an effective collapse who carry oleothorax for years without any difficulty, with disappearance of pus. It is very much simpler for the patient, and I think that it ought to be considered by the surgeons in this particular type of unexpandable lung with pus.

DR. ADRIAN V. S. LAMBERT, New York City.—Before this discussion is closed, I should like to draw attention to one fact which I cannot see omitted in this meet-

ing since, in my opinion, it is the most important fact about tuberculous empyema. You who are doing pneumothorax apparently take the subject lightly and, without any shame at all, blithely tell in how many cases you get fluid and empyema following pneumothorax. Everybody gets it. That is to me a pathetic thing because that is the disease we are talking about, and you are giving it to the patient.

You should question whether or not the pneumothorax is being maintained a little too long in certain cases, whether that is not the reason the empyema develops, and whether or not the empyema comes from poor technique. I think you ought to realize that you are giving this patient tuberculosis of the pleura. He did not start with it. He started with tuberculosis of the lungs. You may have cured him of that, which is a very serious disease, but you have been the direct cause for giving him an equally serious disease.

Another thought I should like to leave with you is that, as I understand it, from the very beginning pneumothorax was designed for the treatment of pulmonary tuberculosis, and it was never intended that it be continued year after year for the treatment of tuberculous empyema. We of the American Association for Thoracic Surgery should like to come here with the message that we feel that you are keeping the patients with tuberculous empyemas too long and that you are sending them to us when a desperate situation arises from thoughtlessness in the length of time you maintain pneumothorax, which was never meant for treatment of tuberculosis of the pleura. Pneumothorax was always intended to be used for the treatment of tuberculosis of the lung.

DR. EGLEE.—I should like very much to emphasize Dr. Lambert's remarks. I shall apply my comments particularly to the picture of pneumothorax treatment in a large public clinic which has just been shown to you in figures. The criticism which will probably be raised against this picture is the old cry, why is it that even in our best public institutions the results of pneumothorax treatment compare poorly to those in private practice? My experience in one of the largest pneumothorax clinics of metropolitan New York leads me to the conclusion that, under the conditions with which we have to contend, the results achieved speak rather well for the efficacy of pneumothorax as a means of control of pulmonary tuberculosis.

These results could be much improved upon if pneumothorax clinics were organized and operated in a less haphazard fashion. I most heartily subscribe to the conclusion drawn from this study: "Artificial pneumothorax should be instituted in municipal clinics only where there is an associated hospital, and there should be continued follow-up to termination of care."

The collective efforts put into the running of pneumothorax clinics has grown into enormous proportions. Much of this is wasted, I believe, because there is not sufficient realization of the fact that pneumothorax is not a method of treatment that can be standardized. It cannot be successfully carried on by clinics operating in the manner of filling-stations to an ever changing clientele. Pneumothorax is, and always will remain, a highly individualized method. It brings the expected results only when it is carried out as such. Every effort should therefore be made to secure continuous follow-up of the case by one group or to secure the fullest cooperation of the various groups in the same community in which the patient is likely to shift places.

This study shows the seriousness of persistent effusion and of the unexpanding lung. These are the pitfalls of the advanced and terminal phases of pneumothorax treatment.

We have learned to avoid the pitfalls of the first phase of pneumothorax treatment. We shall do well to remember the empyema which may follow persistent effusion, as well as the unexpanding lung which so often follows empyema treated

too long medically. Obviously we have looked upon these too lightheartedly. Seeing so much of them, we have become accustomed to them and less anxious about them. It is easier to keep aspirating than to make radical decisions. It is easier to keep refilling than to make radical decisions. The dangerous potentialities of these situations should receive more careful consideration in the future. I am heartily in agreement with the proposition that surgical intervention for tuberculous empyema and unexpanded lung is indicated more often and much earlier than has been customary in the past. I should further more strongly emphasize my conviction that there is an optimal period for surgical collapse in most of these cases and that this period is often too brief and is missed all too frequently. Many failures result from having missed that period when the patient's condition best warranted or permitted surgery. It is here that we most often face difficult alternatives. The patient must be first built up for surgery, but the general condition of the patient will not improve until the empyema has been radically treated. Sometimes the operation must be delayed, but more often the operation should not be delayed too long. Each case has its individual peculiarities, and no scheme of treatment can be applied generally. I know of no other situation in which teamwork of the highest quality between medical and surgical attendants is needed more.

I very much agree with the conclusion that septic empyema is a surgical condition. Its urgency as such is not quite that of an acute appendix, for it is measured in days rather than in hours. The seriousness of septic empyema, arising as it so often does in a patient after a long siege of illness, is reflected in the mortality figures that you have just seen.

THE DIFFERENTIATION BETWEEN ACUTE PUTRID AND NONPUTRID PULMONARY ABSCESS

Its Significance in Relationship to Therapy

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IN SOME of our articles[4, 8] dealing with acute pulmonary abscess, we drew attention to a number of dissimilarities between putrid and nonputrid lesions. More recently we have been impressed by striking differences in the response of these lesions to various forms of therapy. Since putrid and nonputrid pulmonary abscesses differ so much in their response to treatment, appropriate therapy can be rendered only if the two are differentiated from one another. Such differentiation therefore is not merely of academic interest but becomes of clinical importance.

The difference of opinion to be found in the literature concerning the efficacy of various forms of therapy of pulmonary abscess appears to be due, in part at least, to the inclusion of both putrid and nonputrid cases in certain articles. For example, certain authors have maintained that the vast majority of patients with acute pulmonary abscess recover spontaneously or under various forms of nonsurgical therapy. It also has been stated that, in the acute stage of pulmonary abscess, operation is dangerous because infection often is spread by the trauma of traversing acutely inflamed pulmonary tissue which surrounds the abscess. In the case of acute *nonputrid* pulmonary abscess, we are substantially in agreement with both of these views. In the case of acute *putrid* abscess, however, our experience leads us to disagree emphatically. Thus, at most, one-third of the cases of acute putrid abscess, observed by us in the hospital, subsided under conservative forms of therapy. Furthermore, in not a single instance in our series of 118 operative cases of acute *putrid* abscess was there evidence that operation induced a spread of infection regardless of how early in the course of infection operation was performed. Accordingly, we are of the opinion that, if lesions of the putrid and nonputrid variety were carefully differentiated from each other, such widely divergent views might not exist. Our purpose in this communication, therefore, is to emphasize the importance of such differentiation in the hope that more general agreement in regard to certain aspects of acute pulmonary abscess will result and that more effective therapy may be rendered.

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The organisms chiefly responsible for the initiation of putrid abscess apparently are anaerobes. Nonputrid abscess, on the other hand, is initiated by aerobic organisms. The latter commonly include the pneumococcus, *Streptococcus hemolyticus,* and *Streptococcus aureus,* and occasionally the *Streptococcus viridans, B. influenzae,* and Friedländer's bacillus. The relationship of bacteriology to therapy appears significant and will be discussed later.

Another important difference between putrid and nonputrid pulmonary abscess is concerned with the pathologic changes produced in the lung. Putrid abscess begins as a localized area of gangrenous pneumonitis which results presumably from the aspiration of infected particulate matter from the mouth or upper respiratory passages. The aspirated material becomes arrested in one of the smaller bronchi or bronchioles which are situated in the peripheral portions of one of the pulmonary lobes. The resulting area of gangrenous pneumonitis therefore is located superficially within the lobe. Rapid necrosis and liquefaction take place, as evidenced by the fact that the formation of cavities often can be recognized roentgenographically within a week of the onset of the infection. Because of the peripheral situation of the cavity, an intense, overlying pleuritis is produced, which results in local agglutination of the lobe to the thoracic parietes (or occasionally to an adjacent lobe, the diaphragm, or the mediastinal pleura). Since necrosis of the original area of gangrenous pneumonitis is quite complete, a putrid abscess usually consists of a unilocular cavity of substantial size surrounded by only a narrow zone or "shell" of infiltrated lung. This "shell" consists of inflamed, atelectatic, essentially avascular, pulmonary tissue.

A nonputrid pulmonary abscess begins as an area of bronchopneumonia which roentgenographically is not distinguishable from the usual type of bronchopneumonia. Within a variable period of time, however, the frankly purulent character of the sputum indicates that the pneumonic process is of the type which we have designated as "suppurative" or "necrosuppurative."[4] This variety of pneumonic lesion usually is more extensive and diffuse than the localized gangrenous pneumonitis which precedes putrid pulmonary abscess. Within the area of involvement, irregular foci of suppuration and necrosis appear. These often coalesce to form irregular, multilocular cavities. Since the aerobic organisms of nonputrid abscess presumably are less lytic than the anaerobes found in putrid abscess, liquefaction is not as rapid or as complete. Therefore, cavitation is likely to occur more slowly, and the abscess often is surrounded by an extensive area of necrosuppurative bronchopneumonia which fails to undergo complete necrosis. Occasionally, however, the original area of necrosuppurative bronchopneumonia is limited in extent and undergoes fairly complete necrosis.

This results in a lesion quite similar to that noted in putrid pulmonary abscess, *i.e.*, a unilocular cavity surrounded by minimal pulmonary infiltration.

One of the striking differences between acute putrid and acute nonputrid abscess lies in the strict localization of putrid abscess to one part of the lung in the vast majority of cases. In cases of nonputrid abscess on the other hand, the bronchopneumonic process may involve various parts of the lung and often the opposite lung, concomitantly or in sequence. These additional areas also may undergo cavitation completely, partially, or not at all, thus producing multiple cavities or various combinations of cavity formation and pneumonitis.

Whereas putrid abscess may be considered to be a solitary lesion surrounded by a shell of infiltrated lung, nonputrid abscess presents itself in one of four forms as follows: (1) Pulmonary excavation in the midst of, and an incidental part of, an extensive area of necrosuppurative bronchopneumonia, (2) pulmonary excavation as the prominent or predominating lesion, but surrounded by a considerable area of necrosuppurative bronchopneumonia, (3) "typical" pulmonary abscess surrounded by a narrow zone of pulmonary infiltration, similar to the putrid variety, and (4) multiple scattered lesions of the three types just enumerated existing in various combinations.

The actual recognition of putrid and nonputrid abscesses as a rule is not difficult. Their differentiation depends essentially upon the presence or absence of the characteristic fetid odor of the breath or sputum. It is to be emphasized that in the absence of fetid sputum careful attention must be paid to the breath. Thus, in cases of putrid abscess in which spontaneous drainage of the lesion by way of the bronchial tree is not free, foul odor of the sputum may be entirely absent, may be detected only periodically, or may be missed completely by the observer and reported only by the patient. In such cases foul odor may be detected at times, as the patient is instructed to cough forcefully. If uncertainty still exists after a period of observation of several days, diagnostic bronchoscopy may be indicated. In carrying out the latter procedure, it is essential that the instrument be passed sufficiently far into the bronchial tree to visualize the branch bronchus from which pus is issuing. Under such circumstances foul odor often will be detected through the bronchoscope even though both the breath and sputum appear odorless. If pus is present in substantial quantity and is odorless, the diagnosis of nonputrid abscess is warranted. If the amount of pus which issues from the involved bronchus is small and odor is not detectable, another diagnostic bronchoscopy should be performed within a few days.

After it has been determined that an acute pulmonary abscess is of the putrid or nonputrid variety, attention is directed to the subject of treatment. It may be stated at the outset that acute nonputrid pul-

monary abscess demonstrates a much greater tendency to subside spontaneously or under conservative forms of therapy than does acute putrid abscess. This applies to all nonputrid lesions, including the well-localized form (Type 3) which closely resembles putrid pulmonary abscess. In searching for an explanation of the difference in behavior particularly between Type 3 nonputrid abscess and putrid abscess, a consideration of the bacteriology of the two lesions perhaps offers some clue. In both the putrid and the nonputrid abscesses of the type under discussion, some spontaneous drainage of the abscess usually takes place by way of the bronchial tree. This is indicated in the roentgen film by the presence of a cavity containing a fluid level. In nonputrid abscess such drainage very often proves effective in producing cure. In putrid abscess on the other hand, it often fails because drainage alone is insufficient to produce cure, and a second factor, namely, adequate aeration, is essential in order to overcome the anaerobic infection. The principle of providing adequate aeration (oxygenation) applies no less in cases of putrid pulmonary abscess than in cases of anaerobic infection elsewhere in the body. Thus we believe that the lower incidence of spontaneous subsidence of putrid abscess as compared with nonputrid abscess is due to the fact that oxygenation adequate for cure often is not provided even though drainage itself may be fairly good. Accordingly, we believe that, if subsidence of a putrid abscess is to occur under nonoperative forms of therapy, a more free communication between the abscess cavity and the bronchial tree must exist than in the case of nonputrid abscess. A second reason for the much lower incidence of cure in putrid abscess appears to be its failure to respond to medicinal therapy. Thus, the organisms responsible for putrid abscess are little, if at all, influenced by drugs of the sulfonamide group. The opposite appears true in not a few cases of nonputrid pulmonary suppuration. Thus, from the foregoing it may be stated that Type 3 nonputrid abscess, although quite similar to putrid abscess from the viewpoint of pathology, responds more favorably to medical and expectant treatment. Chemotherapy and other forms of conservative treatment to be mentioned later, therefore, in general, are to be recommended in cases of nonputrid abscess.

Occasionally a patient with Type 3 nonputrid abscess presents evidence of severe infection, i.e., high fever, rapid pulse, prostration, etc. Roentgenography may reveal evidence of a rapidly enlarging lesion, a lesion which remains stationary for weeks, a lesion in which perforation into the pleura appears imminent, or one in which perforation has actually occurred. Under any of these circumstances the wisdom of continuing conservative therapy is to be questioned and consideration must be given to surgical treatment. Whether or not surgery will be employed depends on the further course of the disease, except in the cases in which actual perforation into the pleura has occurred. Under

the latter circumstances surgical treatment is indicated without delay. Barring the actual existence of perforation, however, a continuation of conservative therapy is indicated until there is evidence that such treatment is ineffective or unsafe. Our experience indicates that surgery, when employed in Type 3 cases, yields excellent results which are similar to those noted regularly following operative treatment of acute putrid abscess.

In cases of acute nonputrid pulmonary abscess in which pulmonary excavation is surrounded by an extensive area of necrosuppurative bronchopneumonia (Types 1 and 2), nonsurgical treatment is recommended. As was indicated in previous paragraphs, cavity formation in such cases may involve any portion of the original pneumonic lesion. Thus, such cavities often are not situated as superficially as putrid abscesses. When the cavity is situated deeply within the lung, the surrounding pulmonary parenchyma usually is actively and widely inflamed. Overlying pleural adhesions, although usually present, are likely to be more delicate than when the cavity lies superficially. Such cases not only do not lend themselves readily to surgical treatment, but often are influenced unfavorably by operation. Thus, even if the abscess cavity is safely entered and drained, operation not only has little or no favorable effect upon the surrounding pneumonitis but often causes it to spread. Early in our experience we operated upon several patients with nonputrid abscess of Types 1 and 2 of our classification.[4] Some were not benefited, and others were made distinctly worse. For reasons already stated, patients with multiple lesions (Type 4) likewise fall into the nonsurgical category. Patients with nonputrid abscess of Type 1, 2, or 4, complicated by pleural infection should be subjected to operation although in such cases drainage of the pleural infection may have little effect upon the course of the pulmonary lesion.

The conservative treatment of patients with nonperforated abscess of Type 1, 2, 3, or 4 consists essentially of the administration of sulfanilamide, or of one of its derivatives, and the use of repeated transfusions employed for their supportive effect. At times it is surprising to note the remarkable subsidence of even very large lesions, especially in children. We have not been impressed with the value of therapeutic bronchoscopy and do not employ it. Likewise we do not favor the use of postural drainage for the reason that we believe it to be unnecessary. Thus, if an adequate communication exists between the abscess cavity and the bronchial tree, the flow of pus into the latter will stimulate the cough reflex sufficiently to result in expectoration. On the other hand, if an adequate communication is not present, postural drainage and bronchoscopy appear to be of no value.

Unfortunately, nonputrid lesions of Types 1, 2, and 4 at times demonstrate a tendency to spread in spite of treatment. Under such circum-

stances, in the present state of our knowledge, there appears to be little that can be done, and some of the cases go on to fatal termination. In cases of Type 3 on the other hand, surgery, as previously stated, may prove curative.

We turn now to a brief discussion of the therapy of acute putrid pulmonary abscess. As we have already stated, only about one-third of the cases which we observed in the hospital subsided under conservative measures in the acute phase. Those lesions which did not subside during this period (within six weeks of onset) were subjected to operation because experience previously had demonstrated that, if subsidence did not occur during the acute phase, the chances of subsidence under nonoperative therapy in the subacute and chronic phases became increasingly remote. Although some of the patients with putrid abscess upon whom we operated in the acute phase might have recovered subsequently without operation, it was our judgment that in them the continuation of medical treatment was attended with more uncertainty and risk than was surgical treatment. In this connection, it is proper to point out that the goal of treatment is cure in the acute phase in order to avoid the well-known high morbidity and mortality which attends the chronic phase of the disease. Because of lack of space our indications for surgical treatment and a description of the operative technique which we employ in cases of acute putrid abscess cannot be given. References to them will be found, however, in the appended bibliography. The results of early surgical treatment, which also have already been reported, have been more than gratifying not only because of the high incidence of permanent cure but also because of the low mortality.

The importance of differentiating acute putrid from nonputrid abscesses thus is based upon our concept that acute putrid abscess is essentially a surgical disease whereas acute nonputrid abscess is essentially a nonsurgical condition. This statement, as we have already shown, does not imply that we advocate surgical treatment in all cases of putrid abscess and nonoperative treatment in all cases of nonputrid abscess. It does, however, indicate our trend in the general plan of treatment based upon personal experiences over a number of years.

The evidence indicates that a selective therapy is clearly defined when based upon a careful differentiation between putrid abscess and the different varieties of nonputrid abscess. With such differentiation the morbidity and mortality of acute pulmonary abscess should be reduced and the incidence of satisfactory results increased.

SUMMARY AND CONCLUSIONS

Acute putrid pulmonary abscess is essentially a unilocular lesion situated superficially within one of the pulmonary lobes and surrounded by a narrow zone of pulmonary infiltration.

Acute nonputrid pulmonary abscess may assume one of the following four forms: (1) Excavation in the midst of, and an incidental part of, an extensive area of necrosuppurative bronchopneumonia, (2) excavation as the prominent or predominating lesion, but surrounded by a considerable area of necrosuppurative bronchopneumonia, (3) "typical" pulmonary abscess surrounded by a narrow rim of pulmonary infiltration (similar to that noted in cases of putrid abscess), and (4) multiple scattered lesions of the three types enumerated, existing in various combinations.

Acute putrid pulmonary abscess appears to be initiated by a group of anaerobic organisms, whereas nonputrid abscess is produced by aerobes.

Acute putrid pulmonary abscess subsides much less frequently under conservative treatment than does acute nonputrid abscess of Type 3 which resembles it pathologically. Possible reasons for the difference are suggested in the text.

The treatment of acute putrid abscess is essentially surgical, while that of acute nonputrid abscess of Type 3 is essentially nonsurgical.

Acute nonputrid pulmonary abscesses of Types 1, 2, and 4 exhibit the same tendency to respond to conservative measures as does Type 3 and therefore should be treated by these methods. In certain cases, however, such lesions fail to respond to conservative therapy. Experience has indicated that under such circumstances surgical therapy not only is of no value but may prove harmful.

Surgical therapy is indicated in nonputrid abscess of all types when the lesion is complicated by perforation into the pleura. Occasionally operation also may be indicated in nonperforated Type 3 abscess when the clinical manifestations are severe and persistent and the roentgenologic evidence supports the clinical impression of a progressing lesion.

The differentiation between acute putrid and nonputrid abscesses thus forms an important basis for the application of appropriate therapy.

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THE INCIDENCE OF EMPYEMA FOLLOWING INTRAPLEURAL PNEUMONOLYSIS

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THE almost universal acceptance of internal pneumonolysis, introduced by Jacobaeus, and its recent modifications is evidence of its value in the collapse therapy of pulmonary tuberculosis. However, as with all other surgical procedures, risks of possible complications from the operation itself must be weighed against the results which may be obtained. A choice of treatment should then be made which is apt to be the most effective, and at the same time, the least dangerous to the patient.

The purpose of this report is to compare the incidence of tuberculous empyema, the most frequent serious complication of intrapleural pneumonolysis, as it occurs following each of these procedures: 1. Thoracoscopy, with no adhesions cut; 2. Complete pneumonolysis, in which all adhesions on which division was attempted were divided completely; 3. Partial pneumonolysis, in which one or more adhesions were cut only partially through their transverse diameter. The three groups are comparable on the basis of other factors; namely, age, color, sex, and duration of the pneumothorax before operation (average, seven months).

The material is composed of 118 operations performed upon 106 patients between 1937 and 1940 by Moses and Albert Behrend of Philadelphia. The electrosurgical technique with the two-cannula instrument of Coryllos or Bethune was used in every case. All adhesions were divided as near to their parietal attachment as possible. No enucleations were done; and no patients were accepted who showed purulent fluid before operation.

Two terms having various interpretations require definition. An "effusion" is considered to be any collection of pleural fluid detected fluoroscopically, even though transient. This may account for the high proportion of effusions in our cases before, as well as after, operation. Small transient effusions are often regarded as inconsequential in series reported having a lower incidence. The fluid was considered to constitute an "empyema" if it was grossly purulent. The presence of tubercle bacilli or leucocytes was not thought sufficient to warrant the diagnosis of empyema.

A comparison of the figures in Table I illustrates a definite increase in fluid production with increased pleural manipulation. Inasmuch as

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this may be a factor in the formation of empyema, these results are presented here. Postoperative formation of fluid, or increase of fluid already present before operation, occurred most often in the cases in which adhesions were only partially severed, less frequently when adhesions were completely cut, and least of all when thoracoscopy alone was done. The increased incidence of effusion in the partial pneumonolysis group may be explained in some cases on the basis of irritation rather than infection. More manipulation and more prolonged exposure to heat and the rapid exchange of atmospheric air through the open cannulas occur during the cutting of adhesions than during inspection.

TABLE I

	NO. OF CASES	EFFUSIONS BEFORE OPERATION	EFFUSIONS AFTER OPERATION	EMPYEMA
Thoracoscopy	30	13 (43%)	20 (55%)	0
Complete pneumonolysis	54	20 (37%)	34 (64%)	5 (9.3%)
Partial pneumonolysis	34	7 (21%)	28 (82%)	7 (20.6%)
Total	118	40 (34%)	82 (70%)	12 (10%)

The figures on empyema production are most significant. No infection occurred in this series of cases when thoracoscopy alone was done. The incidence of empyema was 9.3 per cent when all attempts at cutting adhesions were completed. However, one of every five cases (20.6 per cent) in which one or more adhesions were partially cut developed empyema. Cutting diseased pulmonary tissue is not the sole cause of empyema. It may be due to spread of infection from the lymphatics and pleural tubercles. However, when it results soon after internal pneumonolysis, it is practically always due to cutting diseased tissue at the time of operation.

CONCLUSIONS

Thoracoscopy is safe and was not complicated by empyema in our series.

If inspection indicates that an adhesion can be completely divided, it should be cut. Complete division of adhesions was followed by empyema in 9 per cent of our cases, but the probability of securing an effective collapse, other factors permitting, outweighs the risks involved.

If lung tissue is accidentally entered during the performance of pneumonolysis, it may sometimes be safer to cut completely through the adhesion, so that retraction and contraction of the stump may seal the opening. Incomplete division of the tissue may permit infection of the pleural space from the exposed area.

Partial division of adhesions carries an incidence of empyema high enough to place the operation in the class of the most serious surgery.

A mortality of 10 per cent is to be expected from this procedure, since 20 per cent of patients are apt to develop tuberculous empyema, which in itself is recognized to have a mortality rate of nearly 50 per cent.[1] Partial division of adhesions is also less likely to produce an effective collapse than complete pneumonolysis. It is suggested, therefore, that no attempt be made to cut an adhesion unless there is reasonable certainty that the adhesion can be completely divided. Other collapse operations should be substituted wherever possible, because they all have a lower mortality.

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CHYLOTHORAX

Report of Case Arrested by Phrenicotomy

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CHYLOTHORAX is a relatively rare condition, there being eighty-four cases reported up to 1937. Reviews of the literature and of cases have been published by Zesas[1] in 1912, Van Nuys[2] in 1931, Mouchet[3] in 1933, and Strauss[4] in 1936.

This condition, although usually found on the right side (because of the right thoracic location of the duct over the major part of its course), may occur on the left side, particularly in injuries to the cervical or upper thoracic portions of the duct. Furthermore, it may be bilateral, or it may be associated with chylopericardium and chyloperitoneum.

According to the anatomic studies of Davis,[5] the thoracic duct is far from being a constant anatomic structure. Its origin at the cysterna chyli on the body of the second lumbar vertebra, its entrance into the right thorax through the aortic hiatus of the diaphragm, and its union with the left subclavian vein after crossing the fourth thoracic vertebra were observed by Davis in 66 per cent of the subjects studied. In 27 per cent of his dissections he observed bilateral thoracic ducts uniting, in the left upper thoracic region, into a single canal, which continued its course to the left subclavian vein in the usual way. The further absence of uniformity of structure in these two broad types is important in an understanding of the various clinical manifestations of chylothorax.

Certain characteristics of chylous fluid differentiate it from pseudochylous fluid as shown chiefly by Wallis and Schölberg.[6] They are listed in Table I.

The chief cause of chylothorax is trauma of either open or closed type. In the latter, fractures of the vertebrae or ribs play important roles. Hyperextension of the spine should be stressed as an important mechanism in the production of thoracic duct injuries. The writers agree with the opinion recently stated by Brown[7] that during this violent action the right crus of the diaphragm with its sharp tendinous edge is capable of a shearing effect on the thoracic duct.

Other etiological factors as outlined by Macnab and Scarlett[8] are: New growths and granulomata (carcinoma, lymphosarcoma, Hodgkins—usually secondary but possibly primary), thrombosis of the left subclavian vein, perforating lymphangitis, aneurysm of the duct, cirrhosis of the

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liver, and filaria. Spontaneous rupture is a possibility in view of the absence of any known factor in several reported cases.

TABLE I

NO.	CHYLOUS FLUID	PSEUDOCHYLOUS FLUID
1	Tends to accumulate very rapidly, and in consequence large volumes are removed at paracentesis.	Collects more slowly, the volume of the fluid varying with the exciting pathologic condition.
2	Generally yellow-white and less perfectly emulsified.	In color a pure milky white solution in the form of an almost perfect emulsion.
3	Degree of opalescence more or less constant at successive tappings.	Opacity generally increases or diminishes at successive tappings.
4	Possesses an odor corresponding to the odor of the food ingested.	Odorless.
5	Microscopically the fluid contains fine fat globules but very few cellular elements.	Microscopically the quantity of free fat is variable; often numerous fine, highly refractile granules are present, and these do not give the reactions for fat. Cellular elements may be numerous and often contain fat; sometimes very scanty.
6	Generally shows a distinct creamy layer on standing.	A cream may or may not form but does not affect the opalescence; a sediment frequently settles out.
7	Specific gravity generally exceeds 1.012.	Specific gravity less than 1.012.
8	Depression of freezing point about –0.51° C., and approximating that for chyle.	Depression of freezing point ranges from –0.56° C. to –0.61° C., and thus corresponds to the figures for blood serum.
9	Total solids vary considerably, but usually greater than 4 per cent.	Total solids rarely exceed 2 per cent.
10	The total protein content generally exceeds 3 Gm. per cent, and, of this, the serum-albumin is the largest fraction; globulin occurs only in traces.	The protein constituents vary between 1 and 3 Gm. per cent, and of these in appreciable quantities.
11	Mucinoid substances absent.	Mucinoid sometimes present.
12	The fat content is generally high, varying from 0.4 to 4 per cent. The fat corresponds in all its properties to the fat contained in food.	The fat content is generally low and may be present in traces only; in its melting point and chemical composition it proves to be pathologic fat.
13	Of the lipines, cholesterin is invariably found, and lecithin occurs in traces only.	The most characteristic lipine is lecithin, though cholesterin is occasionally present.
14	No evidence of the presence of a lipine-globulin combination has been given by previous observers.	The lecithin is mainly combined with the globulin and, when present, is the cause of the opalescence of the fluid. Such fluids resist putrefaction.
15	The salts and the organic substances present approximate the values found for chyle obtained from the thoracic duct.	The salts and organic materials correspond closely to lymph and serous fluids.

In 1933 Mouchet[3] produced experimental chylothorax in dogs by section of the cervical portion and rupture of the pleural dome. Blalock, Cunningham, and Robinson[9] obtained chylothorax in dogs and cats by

ligation of the superior vena cava. The failure to obtain chylous effusion by multiple ligation of the thoracic duct in various regions in the hands of both groups of investigators points to the unimportance of the intraductal pressure as a factor in perforation. Blalock and co-workers observed a diminution of lymphocytes and disappearance of eosinophiles from the blood in their animals.

Special symptoms of traumatic chylothorax as observed by Mouchet are:

1. *Delay in Onset of Two to Forty Days.*—The lapse of eight years between fracture of the left clavicle and onset of chylothorax in Beatty's[10] case is unquestionably a correct estimate. The delay in the appearance of fluid is undoubtedly due to the length of time required for the initial extravasation, which might be termed "retropleural chyloma," to penetrate the *pleural wall.* That a gross perforation of the pleura is not essential for the diffusion of lymph into the pleural space is attested by autopsy findings and the experimental work of Blalock and his co-workers.[9] These investigators were unable to demonstrate any defect in the pleura in their dogs with chylothorax produced by superior vena cava ligation.

2. *Collapse or Shock.*—This condition is undoubtedly caused by change in intrathoracic pressure because relief is usually immediate upon thoracentesis. Mouchet[3] suggested the possibility of protein shock on the basis of his observations following intrapleural injection of chyle in dogs. This phenomenon cannot play a major role in chylothorax when one considers the immediate disappearance of symptoms upon aspiration.

3. *Inanition, Oliguria, and Thirst.*—These symptoms may occur even after intravenous reinfusion of aspirated chyle. Macnab and Scarlett[8] have suggested the possibility of breakdown of lipid metabolism or of loss of some unknown hormone or enzyme. The loss of weight may be strikingly similar to that observed in malignancy.

The diagnosis is established by aspiration of chyle; its identification, by the criteria referred to above. Pseudochylothorax, empyema, tuberculosis, calcareous effusion, and thoracic tumors present clinical pictures similar to chylothorax.

The treatment of chylothorax is essentially aspiration and, if possible, intravenous reinfusion as suggested by Oeken.[11]

Several special manifestations of thoracic duct perforation suggest certain forms of therapy. In the early stage of effusion, which we have termed "retropleural chyloma," the ideal treatment, as suggested by Brown,[7] is thoracotomy and extrapleural drainage with the object of preventing rupture or diffusion into the pleural space.

When chylothorax is caused by injuries in the neck, such as fracture of the left clavicle with compression of the subclavian vein, correction of the clavicular deformity may be indicated as proposed by Beatty.[10]

Since suture of the duct, in its intrathoracic portion at least, appears impossible and in view of the predominant location of traumatic rupture at the level of the diaphragm, the writer performed phrenicotomy with successful walling off of the stoma by elevation of the diaphragm, consequent to diminished respiration and altered crural sulcus. It is, therefore, suggested that phrenicotomy or, preferably, phrenic nerve crushing should be considered if repeated aspiration and intravenous reinfusion fail to give adequate response.

CASE REPORT

A 56-year-old married Italian stone mason was admitted on July 9, 1929, to the Fourth Medical Service of the Boston City Hospital. He complained of constant, nonradiating midepigastric pain, cough, and moderate dyspnea on exertion of three to four months' duration. His present illness began with a nonproductive cough, followed one month later by midepigastric pain of moderate severity which was not related to meals or exertion. Two weeks before entry he noticed dyspnea on exertion. There were no gastrointestinal, genitourinary or neuromuscular symptoms. He was able to work up to one week before admission. During the present illness there was a loss in weight from 145 to 127 pounds.

The family history revealed no contributory information. The patient was married; his wife was living and well. He had six boys living and well; one sibling died during infancy. There was one miscarriage.

Past History.—The patient was born in Italy and came to the United States at the age of 15. He remembered only one illness fifteen years previous when he was in bed for three months with "rheumatism," characterized by pain limited to the right side of the body, especially the right knee. Recently he had had numerous intravenous injections presumably for syphilis.

The pertinent positive physical findings were normal temperature and pulse, moderate emaciation, signs of fluid in the lower half of the right chest, presystolic and blowing systolic murmurs at the apex, blood pressure 130/55 in the right arm, 95/50 in the left, and capillary pulsation in the finger tips.

Laboratory Data.—The urine was not remarkable. The red cell count was 5,100,000; hemoglobin, 80 per cent; the white blood count, 5,700. Differential count showed 70 per cent polymorphonuclears and 30 per cent lymphocytes. The Kahn and Wassermann reactions were positive. Seven sputum examinations were negative for tubercle bacilli. The vital capacity was 1.2 liters. Electrocardiogram showed normal rhythm of 80 per minute, P-R interval 0.14 sec., QRS 0.08 sec. with slurring and notching in all leads, low amplitude, T waves flat in all leads, 3.5 mm., normal axis.

Clinical Course.—The patient was given digitalis. During the first two weeks the temperature rose to 99° and 100° F. nearly every day. On the ninth hospital day a diagnostic thoracentesis in the posterior part of the right chest yielded 50 c.c. of milky brown fluid. On the following day 1,600 c.c. of the same type of fluid was removed. Fluoroscopy immediately after aspiration showed lessened aeration of the right lung; the right diaphragm was flattened and excursion limited. The costophrenic angle was not clear; the heart was moderately enlarged. There was a questionable tumor in the right mediastinum. Roentgenograms taken on the same day showed diffuse mottling of the entire right chest most marked at the apex. There was no free fluid. The left lung was clear. The impression was malignancy or tuberculosis.

The patient showed reaccumulation of fluid and was aspirated twice more during this admission with yields of 1,600 and 1,800 c.c., respectively. His weight dropped from 128 to 110 pounds during this admission. He was discharged seven and one half weeks after admission with a persistence of cough. The discharge diagnosis was tertiary syphilis, syphilitic heart disease, (?) aortic aneurysm, aortic regurgitation, and pleural effusion with chylous fluid.

The patient was readmitted eight times over a period of eight months for recurrence of chylothorax and received a total of seventeen aspirations amounting to 27.5 liters of chyle. During these admissions the following relative points were observed: Repeated Kahn and Hinton tests were positive; pneumothorax failed to prevent reaccumulation of chylous fluid; administration of 1 Gm. of Scharlach R by mouth resulted in its appearance in the chylous fluid on the following day; thoracoscopy revealed no apparent defect in the thoracic duct; the stools showed a four-plus guaiac reaction with negative gastrointestinal x-ray series and slightly diminished free hydrochloric acid in the stomach.

Eight months after his first admission at the suggestion of Dr. E. D. Churchill a right phrenicotomy was done under local anesthesia.

The patient was again admitted seven months later for cardiac decompensation when he stated that his symptoms caused by chylothorax were completely relieved since phrenicotomy. Roentgenograms of the chest showed thickened pleura with a suggestion of a small amount of fluid. However, two aspirations yielded no fluid. The patient died of cardiac failure ten months after phrenicotomy.

At autopsy, restricted to the chest, the following findings were observed: The left pleural cavity was not remarkable. The right pleural cavity was almost completely obliterated by adhesions of the visceral and parietal pleurae. All the structures in the right chest and mediastinum were removed. Posteriorly in the right pleural cavity the pleurae were not adherent, and in this region there was a collection of 150 c.c. of yellowish, milky, chylous fluid. The thoracic duct was isolated, measuring 3 mm. in diameter. At the level of the eighth thoracic vertebra there was a small opening measuring about 1.5 mm. in diameter. The aorta showed tree-bark roughening. There was no aneurysm.

The pericardial cavity was completely obliterated by adhesions. The external surface of the pericardial sac was also adherent to the right lung and the aorta. The heart was slightly enlarged.

DISCUSSION

In the absence of common etiological factors in this patient's history one is confronted with the possibilities of spontaneous or syphilitic perforation. Syphilitic perforation cannot be invoked in the absence of definite syphilitic pathology in the thoracic duct.

The early roentgenographic appearance of a tumor in the right posterior mediastinum emphasizes again the point made by Brown[7]

of this early manifestation of the disease which we have termed "retropleural chyloma." Although the shadow is not specific for the condition, such a shadow occurring with a history of trauma several days to several months prior should suggest the possibility of perforation of the thoracic duct. In this stage of the process diagnostic aspiration should be carried out and followed by thoracotomy and extrapleural drainage as described by Brown[7] to prevent intrapleural extravasation.

The blood findings showed that there was no decrease in the number of lymphocytes and disappearance of eosinophiles as demonstrated by Blalock and his co-workers[9] in fatal experimental chylothorax. The latter findings perhaps appear in those clinical cases which go on to death. It might be used, therefore, as a prognostic sign in this condition.

One cholesterol determination of the blood carried out three months after the patient's first admission and approximately six months after the onset of symptoms showed a normal value of 200 mg. per cent. The nonprotein nitrogen determined at the same time was normal (24 mg. per cent). This constituent was definitely elevated (68 mg.) during the patient's last admission.

The possibility of coincidental healing of the chylothorax in this case does not appear likely when one considers that chylous fluid appeared to re-form at the same rate up to the time of phrenicotomy. Although autopsy showed an accumulation of 150 c.c. of chyle and persistence of a small opening in the duct at the level of the eighth dorsal vertebra, this chylothorax was healed for practical purposes when one considers that not a single aspiration was necessary for nine months after phrenicotomy as compared with seventeen therapeutic aspirations over a period of eight months prior to this procedure.

The mechanism by which phrenicotomy favors healing of low thoracic duct perforations is presumably due to elongation and narrowing of the crural sulcus with apposition of the diaphragmatic and mediastinal parietal pleurae. Other mechanisms, such as elimination or diminution of the aspirating action on the open thoracic duct of the respiratory movements, must also operate. Finally, it is possible that the limitation of the respiratory movements favors clot formation of lymph by eliminating the defibrinating action ascribed to these movements in case of hemothorax.

The mechanism of death in chylothorax is still unknown. The cures obtained by aspiration and failures in certain cases in which aspiration is combined with intravenous reinfusion raise a paradoxical situation. It is highly probable that anatomic anomalies in the form of dual thoracic ducts, etc., are responsible for many of the cures.

SUMMARY

A case of chylothorax is described in which the perforation occurred at the level of the eighth thoracic vertebra. Positive tests for syphilis and death due to cardiovascular syphilis raise the possibility of syphilis as the cause of perforation in this case.

Chylothorax was arrested by right phrenicotomy.

The mechanism of this procedure with its attendant elongation and narrowing of the crural sulcus together with limitation of respiratory movements is discussed.

There was no alteration in the lymphocytic and eosinophilic elements in the blood.

The authors wish to express their appreciation to Dr. E. D. Churchill for his permission to operate upon this patient and for his helpful suggestions.

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STUDIES ON THE ETIOLOGY OF POSTOPERATIVE PULMONARY COMPLICATIONS*

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DESPITE noteworthy contributions in the past and present medical literature pertaining to the causes and prevention of postoperative pulmonary complications, this disease complex in which are included bronchitis, atelectasis, and pneumonitis continues to be a source of considerable morbidity and a cause of high mortality, particularly in upper abdominal surgery.

All statistical studies on the incidence of postoperative pulmonary complications are in agreement with respect to the relatively high incidence in the hernia-laparotomy group of cases as compared with extra-abdominal types of operations. In King's study,[1] this incidence was 14.3 per cent in the hernia-laparotomy group and 1.2 per cent in all other operations. In our own series,[2] the corresponding percentages were 10.9 and 2.6. The occurrence of pulmonary complications in the extra-abdominal group has received too little emphasis. In this group the mechanical factors which are considered by most authors to be of primary etiological importance (namely, pneumoperitoneum, rigidity of abdominal muscles due to wound pain, restraining dressings, intestinal distention with resultant elevation of the diaphragm) are not in operation. This would suggest the existence of some basic etiological factor or factors common to all types of operation, whether intra- or extra-abdominal; the usually accepted mechanical factors which predispose toward pulmonary hypoventilation become secondary in explaining the preponderantly high incidence in the intra-abdominal group.

The following case focused our attention sharply on this problem and the above implications. A patient, male, aged 28 years, had previously been subjected to an ileostomy as a preliminary to other treatment for chronic ulcerative colitis. He was admitted to the New Haven Hospital, with a prolapse of the proximal ileal loop. Attempts to reduce this through prolonged manual manipulation by the patient and his physician were unsuccessful. A spinal novocain anesthesia of 125 mg. in the third lumbar interspace relaxed the abdomen and reduction was simple. The same evening and the following day the patient had a rise in temperature to 102.6° F., cough, dullness and suppression of breath sounds over the right lower lobe, from which he recovered entirely in two days with symptomatic treatment.

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Factors which are inherent in all operative procedures and which conceivably could be etiological in postoperative pulmonary complications are: (1) tissue trauma and the absorption of the chemical products of cellular disintegration; (2) anesthetic agents, local or general; (3) pre- and postoperative sedation; (4) postural restrictions. This paper is intended to consider these factors individually, with a brief summary of our experimental observations relating thereto.

TISSUE TRAUMA AND THE PRODUCTS OF CELLULAR DISINTEGRATION

Since the work of Best, Dale, Dudley, and Thorpe,[3] it has been known that lung tissue has a high content of histamine or histamine-like extractives, exceeding in proportion to unit weight that of any other tissue by ten or more times. Furthermore, it has been demonstrated that this lung-bound histamine may be liberated in perfusates by the action of various toxins such as snake venom,[4] staphylococcus toxin,[5] and the bronchial insufflation of hydrocarbon and other vapors.[6] The action of liberated histamine locally on the lung should be to induce bronchiolar constriction, bronchial hypersecretion, and pulmonary arterial constriction.[7] These effects in a lung where the respiratory mechanism is already depressed by sedation and various mechanical factors may well lead to lobular and lobar obstruction and its sequelae.

In conjunction with Dr. A. Z. Gilman,[8] a series of experiments was set up to test the effect of operative manipulations on lung histamine. In a group of adult cats under nembutal anesthesia, a biopsy was taken of the right lower lobe through a small thoracotomy incision for purposes of histamine bioassays, following which the animals were subjected to laparotomy and deliberate handling of the intestines. At intervals of twenty-four to forty-eight hours the animals were sacrificed with intravenous nembutal, and assays of the remaining lungs were made. No significant change in the lung histamine levels could be demonstrated. However, apparently spontaneous variations in histamine content from animal to animal were observed. Age of the animal suggested itself as the responsible variant. Assays on the lungs of newborn kittens were therefore made, and it was demonstrated that histamine was either absent, or in the order of 5 gamma* or less per gram of lung tissue (average, 2.4). Litter mates of the sacrificed newborn kittens were allowed to mature naturally for a period of five months; the lung histamine levels rose to an average of 9.2 gamma per gram. Usual adult levels in the cat were found to vary from 15 to 35 gamma. A third set of litter mates was subjected to daily graded parenteral injections of pure histamine hydrochloride solutions in divided doses, finally attaining a maximum dosage of 0.9 to 1.0 mg. per day. These animals matured and gained weight as well as the controls, but at the end of injection periods

*1 gamma = 0.001 mg.

as long as thirty-eight and fifty-six days, the lung histamine content showed no increase beyond that occurring with natural age. It seems justifiable to conclude, therefore, that synthetic histamine, at least, is not eliminated by fixation and storage in the lungs, as some authors have supposed, but probably by the hydrolytic action of histaminase in blood and tissues. Histamine assays have recently been made on autopsy material from human stillborn and newborn infants, and in no case has a level higher than 5 gamma per gram been demonstrated. The age differential in histamine levels so demonstrated suggests a possible correlation with the rare occurrence of postoperative pulmonary complications in infancy and early childhood and their increasing frequency with age.

ANESTHESIA

The type of anesthesia has no well-demonstrated direct relation to the incidence of postoperative pulmonary complications. Beecher[9] showed that following abdominal operations of various types and various anesthesia, the subtidal lung volume (that is to say, the air content of the entire respiratory tract at the end of a normal tidal expiration) suffered a progressive reduction averaging 20 per cent on the fourth day postoperatively, after which it recovered slowly toward preoperative levels through the second week. To supplement these findings, it has seemed important to us to study the effect of a general anesthetic alone before operative intervention complicates the picture. For this purpose, a series of seventeen adult patients with negative respiratory histories and chest findings was studied. Preoperative determinations of the subtidal lung volume were carried out at least in duplicate according to the technique of Christie.[10] The patients were then given preliminary injections of atropine (gr. $\frac{1}{150}$ to $\frac{1}{100}$) and morphine (gr. $\frac{1}{6}$ to $\frac{1}{4}$). Anesthesia was induced with rectal avertin in dosages of 2.2 to 4.0 c.c. per square meter of body surface area. The lung volume determinations were repeated within a few minutes. Fourteen of the seventeen patients showed a decrease in subtidal lung volume, averaging 510 c.c. or 19.5 per cent of their mean subtidal volume. In serial determinations on the same patients, the greatest decline in lung volume occurred twenty-five to forty minutes after administration of the avertin. Two of the three patients who demonstrated no decrease were obviously not sufficiently anesthetized by the drug. Whether this demonstrated that diminution in lung volume is a central depressive effect or the result of peripheral hypotonus in the respiratory musculature is not certain. It is significant that the same effect occurs in natural sleep.[11] This is demonstrated graphically in Fig. 1, which is a spirometer tracing from a patient, aged 17 years, who had been subjected to left lower lobectomy for bronchiectasis sixteen months previously, with an excellent end result. During the course of a routine lung volume checkup on a sultry,

hot afternoon, he became drowsy. As he dozed off, the respirations became shallow and slower, and the expiratory level fell away rapidly, only to rise with each awakening.

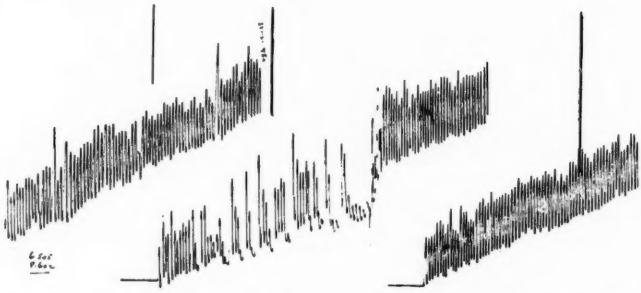

Fig. 1.—Spirometric tracing, showing downward shift in respiratory level with beginning of sleep.

Other effects of avertin administration on the respiratory mechanism were a decrease in oxygen consumption and a decrease in minute respiratory volume.

It appears to be demonstrated therefore that anesthesia with avertin, and probably with any general agent, is attended by an immediate shift in respiratory level in the direction of pulmonary deflation. Since the normal expiratory mechanism is passive, and the expiratory level is determined by a balance between the elastic recoil of the lung and the tonicity of the respiratory musculature, the above results are logically to be expected.

The question now arises whether local anesthesia plus sedation can bring about the same results as general anesthesia under avertin. Experiments are now in progress in this laboratory to answer this query. The effect of sedation (barbital and morphine) has thus far been inconstant and largely negative in normal adults; with large doses of morphine there have been occasional slight decreases in subtidal volume, but not in the order of those seen with avertin. However, determinations of subtidal lung volume before preoperative sedation and immediately after uncomplicated inguinal herniorrhaphy under local procaine anesthesia have demonstrated in all of four cases so studied a comparable reduction in subtidal lung volume with that observed under avertin anesthesia. A further study of operation under local anesthesia is being made,[12] since the results thus far lend support to the suggested humoral theory for the production of postoperative pulmonary complications.

INFECTION

The exudative and obstructive phases of postoperative pulmonary complications are commonly followed by bacterial invasion of the lung.

In 1918, Whipple[13] found that pneumococci could be obtained with great regularity from the sputum of patients afflicted with postoperative pulmonary complications and that the pneumococci were in the conglomerate Type IV. Since the work of Cooper and his associates[14] with further type differentiation of pneumococci, it has seemed desirable to restudy this problem, using direct typing as well as cultural techniques.* In a series of fifty-two postoperative pulmonary complications in which typable pneumococci could be recovered from the sputum, the distribution of types is shown in Table I. It is apparent that the types are widely distributed, which supports the assumption that the pneumococci are secondary invaders from the nasopharyngeal flora.

TABLE I

CLASSIFICATION AND INCIDENCE OF PNEUMOCOCCUS TYPES RECOVERED FROM SPUTUM OF FIFTY-TWO PATIENTS WITH POSTOPERATIVE PULMONARY COMPLICATIONS

TYPE	CASES	TYPE	CASES
III	5	XVII	3
V	1	XVIII	3
VI	6	XIX	3
VII	2	XX	1
VIII	5	XXI	1
IX	2	XXII	1
XI	1	XXIII	4
XIII	2	XXVII	1
XIV	4	XXIX	1
XV	2	XXXI	1
XVI	2	XXXII	1

SUMMARY

A group of clinical and experimental observations relating to the etiology of postoperative pulmonary complications is presented in summary form.

An age differential in the levels of lung histamine has been observed. The failure of parenterally administered histamine to be accumulated in the lung has been pointed out.

A reduction of pulmonary subtidal volume by approximately 20 per cent, due to the immediate effects of general anesthesia (avertin) before operation, has been demonstrated.

A similar immediate reduction in subtidal volume from the effects of operation under local procaine anesthesia with preliminary morphine sedation has been found.

A humoral theory for the etiology of postoperative pulmonary complications is proposed. This assumes the liberation from sites of operation of histamine-like substances which provoke secretion and bronchoconstriction in the lung, thus setting the stage for the action of

*We are grateful to Dr. S. Bayne-Jones and Mrs. Florence Fox Cherman, his technician in surgical bacteriology, for their collaboration with this problem, which will be further investigated.

already well-recognized mechanical factors, which determine secondarily the preponderantly higher incidence in the abdominal type of operation.

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FAT EMBOLISM AS A POSTOPERATIVE COMPLICATION OF EXTRAPLEURAL THORACOPLASTY

Report of Two Proved Cases and Five Suspected Cases

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THE occurrence of fat embolism in the lungs has been reported as a complication of various types of injuries and diseases. Among these are cases of traumatism to the soft tissues, fractures of the long bones; operations of many types, including those on bones, diseases such as diabetes, tuberculosis, and renal diseases, and finally, cases in which no cause for the emboli could be ascertained. There seems to be little doubt that mechanical disturbance of fat in any fat depot is uniformly followed by the appearance of intravascular fat in varying amounts. It has been shown, however, that traumatism is not the sole cause for the appearance of fat embolism. In support of this statement many instances have been reported of fat embolism following burns, cardiovascular-renal disease, pneumonia, poisoning by phosphorus, carbon monoxide, and chloroform, and profound sepsis.

Within the past two and one-half years, two patients upon whom we had performed thoracoplasty died, and at autopsy fat emboli were found scattered densely throughout both lungs and these emboli were thought to be the cause of death in each instance. It was felt that these cases were of sufficient interest to report, for two reasons: first, because as far as we can ascertain no other instances of fat embolism following thoracoplasty have been reported; and second, because of the fact that fat embolism may well be the cause for the dyspnea, cyanosis, and mental changes that are noted in some patients following thoracoplasty. After having seen at autopsy the two cases herewith reported, and having followed them clinically, we have reviewed the hospital charts of the last 100 cases of thoracoplasty in the hope of finding additional cases. We have discovered five such cases in which the signs and symptoms during convalescence suggested strongly the patients' having had lesser degrees of pulmonary fat embolism from which they recovered. We cannot prove the presence of fat embolism in these five cases, but we hope to be able to do so in subsequent cases in which the clinical course is suggestive of fat embolism.

From a mechanical viewpoint, the operation of thoracoplasty offers an excellent set of conditions under which fat embolism can take place.

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Each rib which is resected must be divided in two places, and often additional short segments of this rib are removed with one or two bites of a rongeur. The removal of the transverse processes of the vertebra involves the section of a wide bony surface and leaves a considerable expanse of cut, cancellous bone in its wake. This extensive operative traumatism to bone furnishes the fat (from the marrow) and the open-mouthed vessels (from the cut edge of the bone), a combination which is ideal for the production of fat embolism. An additional fact of importance is that the operative site is very near the mediastinum where the negative pressure in the thorax acts more forcibly on the veins in the operative area. We have noted on inspection, just as many other surgeons must have, that the bleeding from the cut ends of proximal rib stumps or transverse processes ceases or becomes appreciably less on inspiration. Also, the thoracoplasty incision is quite long and involves a considerable amount of subcutaneous fat, and this is likewise exposed to traumatism when the scapula is being raised from the chest wall.

REPORTS OF TWO PROVED CASES OF FAT EMBOLISM

CASE 1.—T. C. (U-566 Hamilton County Tuberculosis Sanatorium), a 42-year-old white male, was admitted to the sanatorium on Dec. 8, 1935. He first noticed weakness and loss of strength in July, 1935. About two months after the onset of his symptoms, he complained of a pain in the lower left chest and coughed up small amounts of blood. One month before admission a physician made a diagnosis of pulmonary tuberculosis, put the patient to bed for five weeks, and finally sent him to the sanatorium.

The patient was emaciated, weak and pale, appearing to be about 55 years of age. Aside from the findings in the thorax, there was nothing remarkable on general physical examination. Roentgenograms of the chest revealed caseous bronchopneumonia in the first and second interspace trunks on the right side and a small area of caseous bronchopneumonia in the lower right lobe. There was a moderate sized cavity in the left lung. There was also some caseous bronchopneumonia in the lingual tips on the left. A moderate amount of emphysema was present at both bases. In April, 1936, pneumothorax treatments were begun on the left side, which were continued for one year. The lung could not be completely collapsed because of adhesions in the vicinity of the cavity. At the end of the year, the cavity was still open and the sputum was positive, so a thoracoplasty was decided upon.

On Sept. 7, 1937, the first stage of a thoracoplasty was performed. The operation, done under local anesthesia, consisted of the removal of long segments of the first three ribs. It was well tolerated, the wound healed by first intention, and the convalescence was uneventful.

Five weeks after the first-stage operation, the second stage was performed. Under local anesthesia, long segments of the fourth, fifth, sixth, and seventh ribs were removed from the transverse processes laterally. The transverse processes were not resected. The operation was well tolerated, and no concern was felt about the patient's condition immediately after operation. He vomited several times during the afternoon of the day of operation and did not complain of unusual

pain or dyspnea. The pulse was between 110 and 120 during the day. At 7:00 P.M. on the day of operation his temperature was 101° F., and the pulse rate was 130. He was somewhat apprehensive at this time, but otherwise was no different from the average postoperative thoracoplasty patient. At 11:00 P.M. he had a very profuse diaphoresis and his apprehension had increased, although he was entirely rational and mentally alert. At 4:00 A.M. the next day (about twenty hours after operation) his condition had not changed appreciably. However, within the next hour and one-half his pulse became more rapid, soon being unable to be counted. He became extremely dyspneic and cyanotic and died twenty-two hours after operation.

An autopsy was performed, but permission to examine the head was not granted. There was no evidence of tuberculosis in the abdominal cavity or intestinal tract. Two areas of caseous tuberculosis were found in the spleen. The kidneys were pale pink in color and on section showed poorly demarcated cortical and medullary zones, and numerous whitish and yellow patches throughout. There was a caseous area in the right adrenal gland which was thought to be tuberculous.

The heart was pale and flabby, and all the chambers were slightly dilated. The coronary vessels were thick-walled, intact, and patent.

When the left pleural cavity was opened, two pockets of thick tuberculous pus were encountered, and the lung was found to be densely adherent to the chest wall everywhere. The left lung on section showed a thick-walled cavity in the upper lobe, with much scarring and fibrosis around it. There was some caseous bronchopneumonia in the upper lobe and in the superior portion of the lower lobe. The left lung was "edematous and congested."

The right pleural cavity contained no fluid. The lung was not adherent to the chest wall. On section the right lung showed no evidence of active tuberculosis, although there were several areas of fibrosis. Scattered through the right lung were "several areas of reddening which are friable and elevated, small bits of these areas sink readily in water. These areas are more prominent at the base." The lung was reddened and moist.

The gross diagnosis as to the lungs was early tuberculous lobular pneumonia. However, frozen sections of the lungs stained with scarlet red showed massive fat emboli throughout both lungs.

CASE 2.—R. T. (Y-282 Hamilton County Tuberculosis Sanatorium) was a 33-year-old white woman on whom a positive diagnosis of pulmonary tuberculosis was made in 1935. She improved remarkably on bed rest and remained symptom-free for nearly two years, when she fell ill again. She developed pleurisy on the right side and began to cough. An attempt to induce an artificial pneumothorax failed, and the phrenic nerve was divided on the right. She again was kept in bed at home, where she gained 30 pounds and was improved clinically. However, the cough continued, and sputum remained positive for tubercle bacilli.

She was admitted to the sanatorium in June, 1938, at which time a diagnosis of far-advanced pulmonary tuberculosis was made. She was well nourished and did not seem acutely ill. The general examination was not remarkable, save for a chronic otitis media on the right. X-ray examination of the chest revealed scattered healed lesions in the upper lobe of the left lung. In the right lung there was a large cavity which occupied the lower portion of the upper lobe and a portion of the middle and lower lobes. There was a small pleural effusion on the right, with thickened pleura at the base.

A thoracoplasty on the right side was decided upon since a phrenicotomy had been performed and compression of the lung by artificial pneumothorax was im-

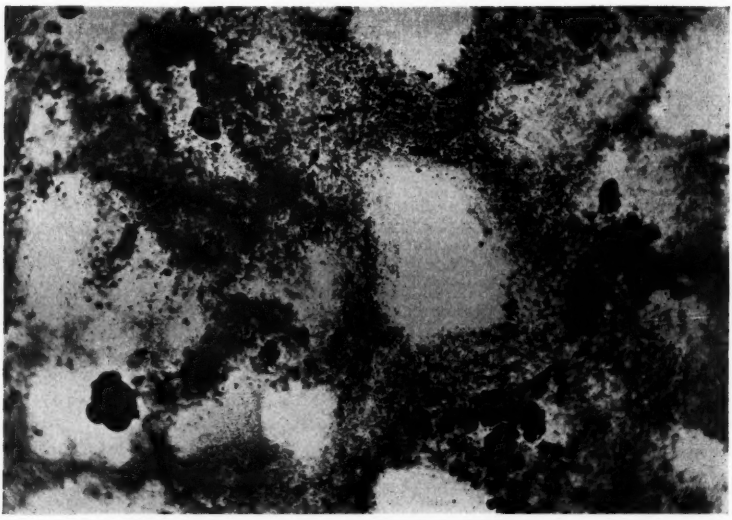

Fig. 1.—Photomicrograph of lung in Case 2. Note the fat emboli in the capillaries.

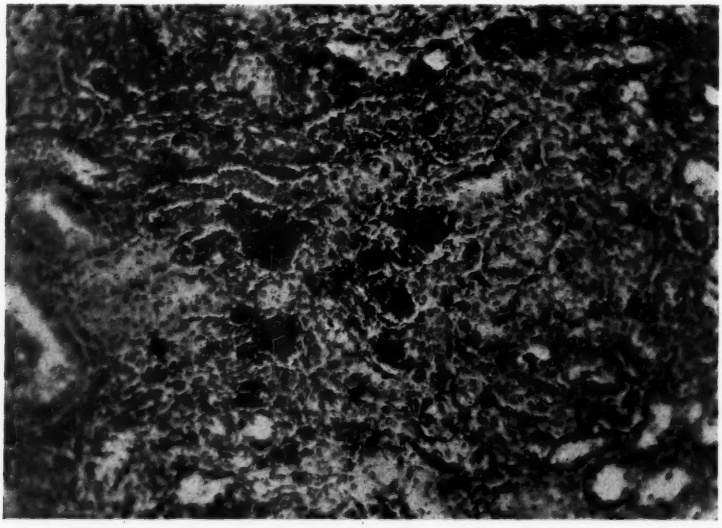

Fig. 2.—Photomicrograph of kidney in Case 2, showing fat emboli in the capillaries.

possible. The cavity lay in the lower portion of the thorax, near the hilum and behind the eighth rib. The thoracoplasty was therefore done from below up, i.e., started at the tenth rib. The first stage was done July 23, 1938, under cyclopro-

pane anesthesia. Long segments of the tenth, ninth, eighth, and seventh ribs were resected from the transverse processes laterally. The convalescence was entirely uneventful.

Seventeen days after the first stage, the second-stage thoracoplasty was performed. Long segments of the sixth, fifth, and fourth ribs, were removed from the transverse processes laterally. There was considerable mobility of the lung, and some respiratory difficulty was encountered at the close of the operation. A tight dressing, however, did away with this respiratory embarrassment. On the second, third, and fifth days after operation she complained of shortness of breath. The attacks of dyspnea were short-lived and never serious. Aside from them, the convalescence was uneventful.

After nine months the sputum was positive, and on x-ray examination it was evident that the cavity was not entirely closed. A third stage of the thoracoplasty was performed, therefore, on May 9, 1939. At this time long segments of the third, second, and first ribs, together with the transverse processes, were resected and 10 per cent formaldehyde solution was applied to the rib beds.

The first ten days of the convalescence were in no way abnormal; the wound healed by first intention, and there was no dyspnea or unusual pain. On the tenth day she complained of dyspnea, which had come on gradually and was unaccompanied by fever. She expressed some anxiety for her welfare, but was alert and keen. On the twelfth day after operation she became gradually much more dyspneic and began to show cyanosis for the first time. A few moist râles were heard at the left base. She was placed in an oxygen tent, with some relief, and was kept there until her death. On the thirteenth day she was confused and irrational, requiring restraint, and a lumbar puncture was done. The spinal fluid was normal. The dyspnea and cyanosis persisted. On the fourteenth day all the symptoms had progressed in severity, and at this time a urinalysis revealed many fat globules in the voided urine. By the fifteenth day she was in coma, very cyanotic and dyspneic, and she died on that day.

An autopsy was performed, but the brain was not examined. Aside from the findings in the lungs, there was nothing of importance to be learned from the examination.

The right pleural cavity was entirely obliterated by adhesions and had been reduced to approximately one-third of its normal size by the thoracoplasty. The pleura was thickened and of a grayish color. The right lung was subcrepitant throughout, but the lower lobe was firmer than the upper and middle lobes and no areas of softening were found. On section the right upper and middle lobes were grayish, subcrepitant, and moderately wet. A few soft and firm gray nodules 1 to 2 mm. in diameter were found scattered throughout these two lobes. A small, firm-walled cavity filled with caseous material was found at the level of the second interspace anteriorly. The right lower lobe on section was generally reddish-black, tough, wet, and airless, with several tiny gray nodules scattered through it.

The left pleural cavity was partially obliterated by adhesions. The pleura over the left lung was thickened and grayish. On palpation the left lung was subcrepitant and firmer than normal. On section both the upper and lower lobes on the left were reddened, wet, and subcrepitant. No nodular or caseous areas were found in the left lung.

Frozen sections from both lungs stained with scarlet red showed discrete and conglomerate globules of fat in the capillaries and smaller vessels (Fig. 1). Many tiny fat globules were found scattered throughout both kidneys (Fig. 2).

REPORTS OF CASES IN WHICH FAT EMBOLISM WAS NOT PROVED, BUT IN WHICH THE SIGNS AND SYMPTOMS IN RETROSPECT WERE SUGGESTIVE OF FAT EMBOLISM

In each of these cases there was no evidence at any time of paradoxical respiration, mediastinal shift, or pneumonia. The dyspnea and cyanosis were not accompanied by fever in any case.

CASE 1.—A. M. (Y-82), a white female, aged 32 years, underwent first-stage thoracoplasty on the right side on Nov. 25, 1938. Posterior segments of the first, second, third, and fourth ribs were removed. The postoperative course was uneventful.

On Dec. 4, 1938, second-stage thoracoplasty was done on the right side. Posterior segments of the fifth, sixth, and seventh ribs were removed. The postoperative course was uneventful for five days, at which time the patient became extremely dyspneic. The pulse was rapid and there was marked sweating. The patient was very apprehensive. She was kept in an oxygen tent for nine days, at the end of which time the dyspnea had ceased.

CASE 2.—M. O. (X-480), a white female, aged 33 years, underwent first-stage thoracoplasty on the left side on May 12, 1938. Posterior segments of the first, second, and third ribs were removed. The postoperative course was uneventful for five days, at which time the patient became very dyspneic and extremely apprehensive. There was profuse sweating, and the pulse was weak and rapid. Normal saline solution, 250 c.c., and 375 c.c. of citrated blood intravenously were given, with little effect. She was placed in an oxygen tent for six days, at the end of which time dyspnea had ceased.

CASE 3.—M. R. (Y-485), a white female, aged 28 years, underwent first-stage thoracoplasty on the right side on Jan. 10, 1939. Posterior segments of the first, second, third, and fourth ribs were resected. The postoperative course was uneventful.

On Jan. 31, 1939, second-stage thoracoplasty was performed on the right side. Posterior segments of the fifth, sixth, and seventh ribs were removed. The postoperative course was smooth for five days, at which time extreme dyspnea accompanied by slight cyanosis appeared. The patient was very apprehensive and perspired freely. Dyspnea persisted for five days, after which it gradually ceased.

CASE 4.—E. S. (Y-468), a white male, aged 39 years, underwent first-stage thoracoplasty on the right side on Aug. 24, 1939. Posterior segments of the first, second, third, and fourth ribs were resected. The postoperative course was uneventful for five days, when the patient became dyspneic, cyanotic, extremely apprehensive, and irrational at times. There was profuse sweating. Many rhonchi were heard over entire left chest. He was placed in an oxygen tent for five days, at the end of which time all symptoms had disappeared.

On Oct. 10, 1939, second-stage thoracoplasty was done on the right side. Posterior segments of the fifth, sixth, and seventh ribs were removed. The postoperative course was uneventful for six days, at which time the patient became dyspneic, cyanotic, and irrational. There was profuse sweating. He was placed in the oxygen tent for five days, at the end of which time he was rational and the dyspnea had ceased.

CASE 5.—M. M. (Z-504), a white female, aged 26 years, underwent first-stage thoracoplasty on the right side on Nov. 20, 1939. Posterior segments of the first,

second, third, and fourth ribs were resected. Convalescence was complicated on the fourth postoperative day by mild dyspnea, slight cyanosis, and apprehension. These manifestations persisted in mild degree for four days. On Dec. 7, 1939 second-stage thoracoplasty was performed on the right side. Anterior segments of the first, second, third, and fourth ribs were removed. The postoperative course was uneventful. On Jan. 4, 1940, third-stage thoracoplasty was done on the right side. Posterior segments of the fifth and sixth ribs were removed. The postoperative course was smooth and uncomplicated for four days, at which time the patient became ill. She perspired profusely, had extreme dyspnea and cyanosis, and was mentally confused and apprehensive. She was placed in an oxygen tent for four days, at the end of which time she had improved remarkably and had only slight dyspnea.

CONGENITAL ATRESIA OF THE ESOPHAGUS WITH TRACHEOESOPHAGEAL FISTULA

Report of Successful Extrapleural Ligation of Fistulous Communication and Cervical Esophagostomy

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THAT the surgical management of cases of congenital atresia of the esophagus with tracheoesophageal fistula is a difficult problem is indicated by the many reports of various unsuccessful operative procedures.

In this most common type of atresia of the esophagus, the upper segment is hypertrophied, dilated and 3 to 4 cm. in length, and terminates blindly just above the bifurcation of the trachea, while the lower segment has a fistulous communication with the trachea usually about 0.5 to 1.0 cm. above the bifurcation or, more rarely, with the bronchus.[10]

The symptoms of choking, cyanosis, and regurgitation due to aspiration of mucus or feedings are characteristic of this anomaly. Death rapidly ensues due to starvation, dehydration, and aspiration pneumonia.[3, 4]

The diagnosis of atresia of the esophagus is readily made on the typical history, obstruction to the passage of a catheter at 10 to 12 cm. from the alveolar margins, and roentgenologic visualization of the blind pouch with instillation of lipiodol or by esophagoscopic examination. The presence of air in the stomach in cases of atresia of the esophagus indicates a fistulous communication with the lungs.[14]

This common type of atresia of the esophagus presents three problems: (1) feeding; (2) management of the fistulous communication of the lower segment of the esophagus with the trachea, (3) care of the blind pouch of the upper esophageal segment. In treatment of these cases, operative procedures have been directed to one or more of these problems.

The following various methods have been attempted or suggested: gastrostomy,[5, 15, 19, 21, 23] jejunostomy,[3, 5, 9, 14, 22] gastrostomy plus jejunostomy,[10] ligation of the cardiac end of the esophagus plus gastrostomy,[23, 6] transthoracic ligation of the fistulous communication with the trachea, with gastrostomy for feeding purposes,[16] closure of the fistula at the junction with the trachea, together with the connection of the

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esophageal segments by a tube[11] or anastomosis of the segments by a modified Murphy button[7] or direct anastomosis[17, 20] and endobronchial stenosis of the fistula plus gastrostomy.[8]

Mixter,[12] in a discussion of the problem, cited cases of nine patients upon whom he had operated over a period of six years in whom he ligated and divided the fistulous communication to the trachea through a right-sided extrapleural approach, bringing out the distal esophagus as a dorsal esophagostomy and placing a catheter in this for feeding. In some of these cases he added a proximal esophagostomy primarily or secondarily since he stated that the overflow of secretion from the blind proximal end of the esophagus was followed by fatal pneumonia. All his cases terminated fatally, two patients living fifteen and thirty days, respectively.

Bohrer,[2] through a left-sided anterior mediastinotomy, severed the esophagus from the trachea at the fistulous junction, closing the tracheal end by ligation and inversion and bringing out the lower portion, thus making a skin fistula through which feedings could be given. This baby died twelve hours after operation.

Heatly[8] reported two cases operated on by W. G. M. Scott[18] in which transection of the upper end of the stomach, with inversion of the stomach, abdominal esophagostomy of the distal esophagus, and gastrostomy, was carried out. These patients survived 34 and 145 days, respectively.

In a previous report[10] I presented two cases of patients who survived ninety-eight and fifty-three days, respectively, following a procedure which attacked the fistula indirectly, namely, exteriorization of the cardiac end of the stomach and esophagus and insertion of a gastrostomy tube distal to the exteriorized segment, for feeding. After further experience with this method, I became convinced that the fistulous communication with the trachea must be attacked directly.

Although re-establishment of the normal continuity of the esophagus by direct anastomosis of the upper and lower segments after ligation of the fistulous communication to the trachea is the ideal procedure,[20] the likelihood of success is remote. Resection and anastomosis of the esophagus in adult cases is notably unsuccessful, and in experimental animals, with few exceptions, the results have been discouraging. This is largely due to the fact that the esophagus lacks a serosal covering which makes primary healing difficult, and leakage of the highly infective esophageal secretions ensues, with resultant mediastinitis.

I have therefore used a procedure which may be divided into more simple stages and which offers a greater chance of success.

DESCRIPTION OF OPERATION

After adequate preoperative preparation by administration of subcutaneous fluids to correct dehydration, a preliminary gastrostomy of

the multiple purse-string type is carried out under local anesthesia, through an upper left rectus incision, with the catheter brought out through a stab wound to the right of the midline. It is important to pass the catheter through the pylorus into the duodenum or jejunum, as

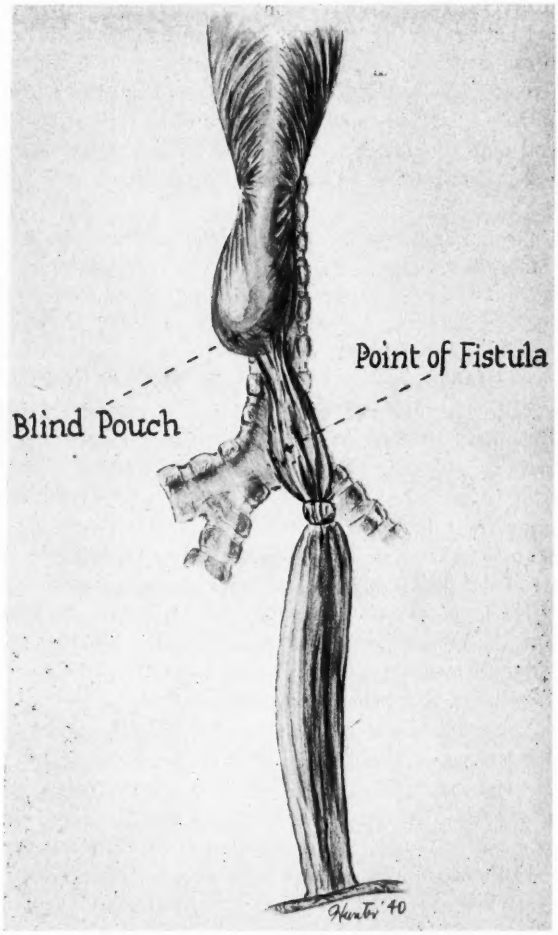

Fig. 1.—Site of ligation of fistulous communication of esophagus to trachea.

suggested by Villemin.[23] If this is not done, spasm of the pylorus, which is induced in these infants by the presence of the catheter in the stomach, will shortly cause leakage of retained gastric contents about the gastrostomy opening. By passing the catheter into the jejunum, these patients have very little difficulty with regurgitation of gastric contents into the trachea if frequent small feedings are used.

After the patient recovers from this procedure, the fistulous communication to the trachea is ligated. The approach used is one which Wangensteen[24] has found to give the best exposure of the esophagus in this area.

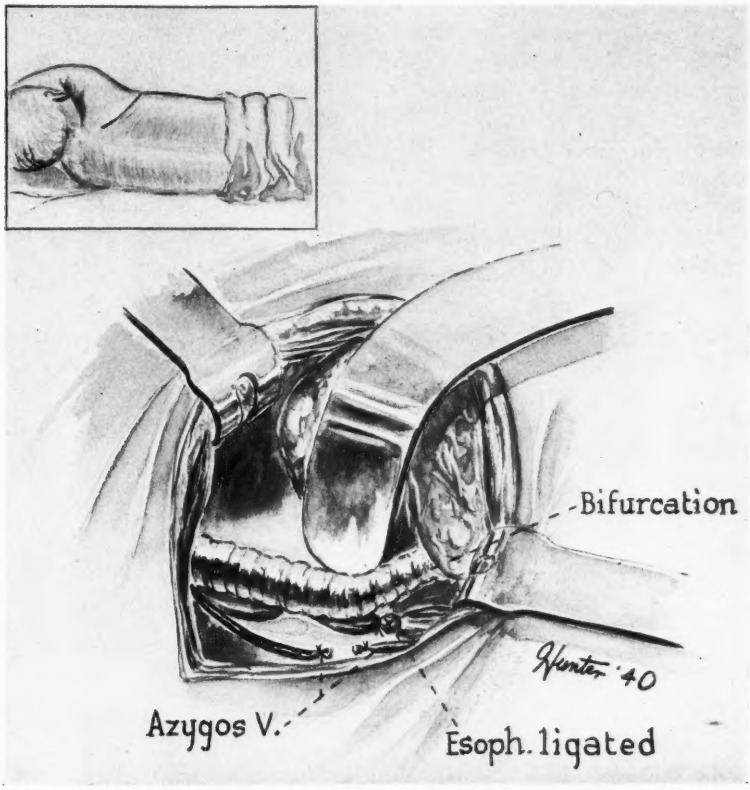

Fig. 2.—Drawing illustrating exposure for extrapleural ligation of fistula.

Through an oblique incision the entire fourth rib on the right side is removed subperiosteally. The periosteal bed of the rib is incised and the pleura stripped from the chest wall. The azygos vein at this level lies along the esophagus at approximately the point of the fistula. The vein is ligated and divided, exposing the esophagus. With care for the vagi, the esophagus is crushed by a forceps and doubly ligated with heavy silk. A soft rubber catheter is left in the extrapleural space and brought out through a stab wound in the fifth intercostal space. The chest is closed tightly.

Cyclopropane anesthesia administered by mask permits collapse of the lung and intermittent expansion by positive pressure. Postopera-

tively suction of 12 mm. Hg is applied to the catheter to assure expansion of the lung and obliteration of the extrapleural space.

The danger of aspiration of mucus from the mouth into the trachea can be taken care of temporarily by frequent aspiration of the mouth with a bulb syringe, making the cervical esophagostomy no emergency procedure.

The third-stage operation consists of mobilization of the upper segment of the esophagus through an incision anterior to the left sternomastoid muscle. The tip of the blind pouch is brought out through a small incision below the left clavicle and opened in three days, completing the cervical esophagostomy. This stage of the procedure is carried out under ether insufflation anesthesia.

At a distant future date an antethoracic esophagoplasty should be performed to establish continuity of the gastrointestinal tract.

REPORT OF CASE

The patient, a 2-day-old male infant, Hospital No. 688745, was admitted to University Hospital on Nov. 28, 1939. The weight at birth was 2,500 Gm.

Fig. 3.—Demonstration of proximal blind pouch of esophagus.

Since birth, feedings had been immediately regurgitated, and large amounts of mucus in the hypopharynx had caused some difficulty in breathing. No attacks of cyanosis had occurred.

Examination showed a well-developed, well-nourished male infant weighing 2,305 Gm. Temperature was 100.2° F. The skin appeared dehydrated and icteric. An excess of mucus was present in the mouth and pharynx. Coarse râles were heard over both lung bases posteriorly. The remainder of the physical examination revealed no other evidence of abnormalities. Passage of a catheter met obstruction 10 cm. from the alveolar margin. Injection of a small amount of lipiodol demonstrated that the proximal esophagus ended in a blind pouch at the level of the third thoracic vertebra (Fig. 3). The lung fields appeared normal, and a large amount of gas in the gastrointestinal tract was noted on x-ray examination (Fig. 4).

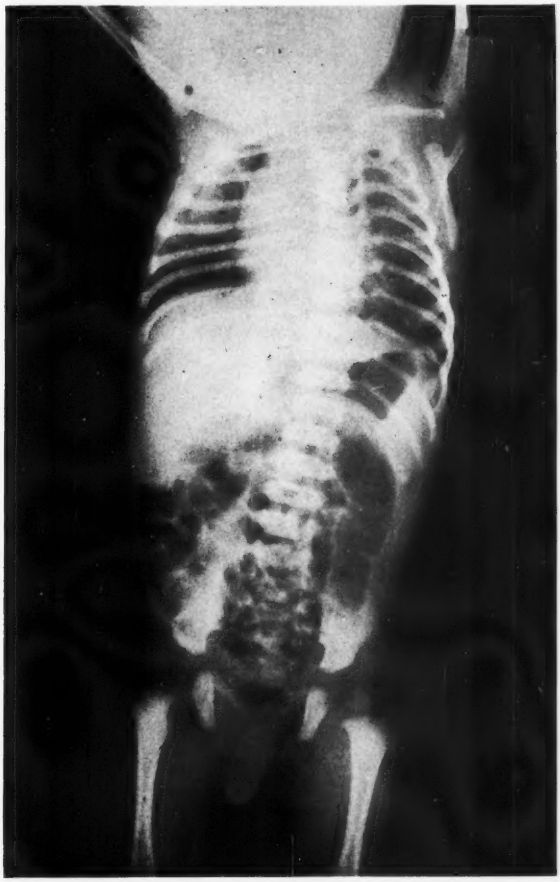

Fig. 4—Roentgenogram showing air in bowel in presence of atresia of esophagus, indicating communication of lower esophagus with air passages.

After correction of the dehydration by the administration of fluids subcutaneously, a gastrostomy was performed under local anesthesia on Nov. 29, 1939, with the catheter passed through the pylorus and well into the jejunum. This procedure was well tolerated, and feedings of 5 c.c. every fifteen minutes were started at once. During the next few weeks occasional attacks of cyanosis occurred, necessitating frequent aspiration of mucus from the mouth.

On Dec. 6, 1939, the fistulous communication of the esophagus to the trachea was identified through a bronchoscope and was coagulated by an electrode passed into the fistula. That this was not successful was indicated by the appearance of methylene blue in the mucus aspirated from the mouth after injection of the dye into the gastrostomy tube.

On Dec. 29, 1939, a second bronchoscopic examination showed marked edema about the site of the fistulous opening in the trachea, and no attempt was made to cauterize the tract.

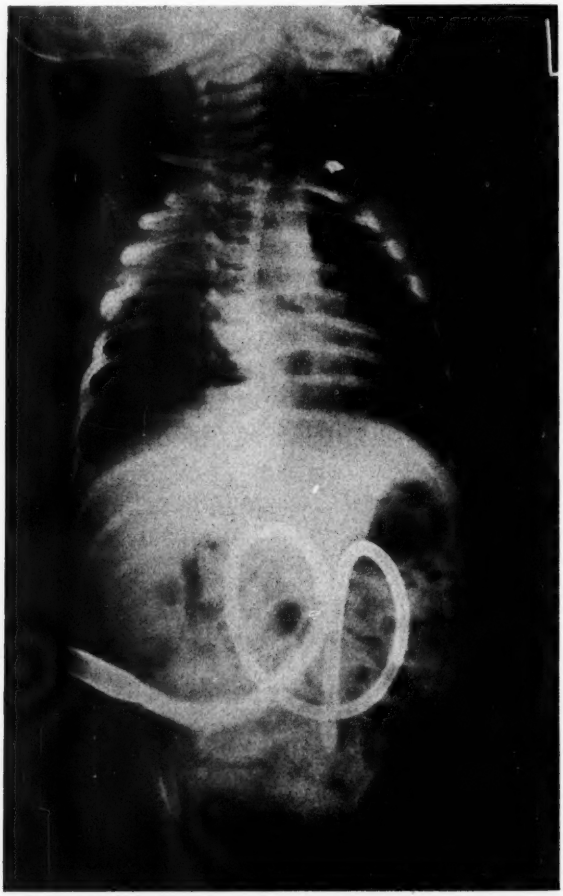

Fig. 5.—Roentgenogram showing resection of the fourth rib on the right side, and the presence of the feeding catheter in the jejunum.

On Jan. 5, 1940, extrapleural ligation of the esophagus at its fistulous communication with the trachea was carried out. A 55-c.c. blood transfusion was given to the baby the day before the operation, and a second 75-c.c. transfusion was given immediately following surgery.

Except for a temperature elevation to 101.8° F. on the day of surgery, the postoperative course was without event. The amount of mucus collecting in the mouth

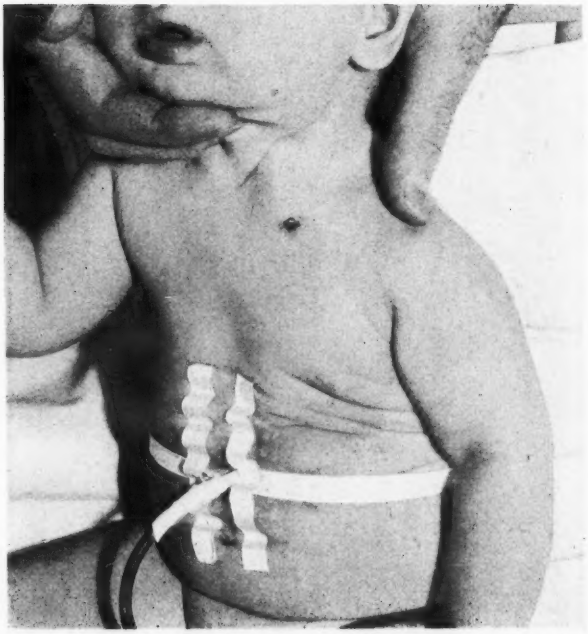

Fig. 6.—Patient at 185 days of age, showing cervical esophagostomy and gastrostomy.

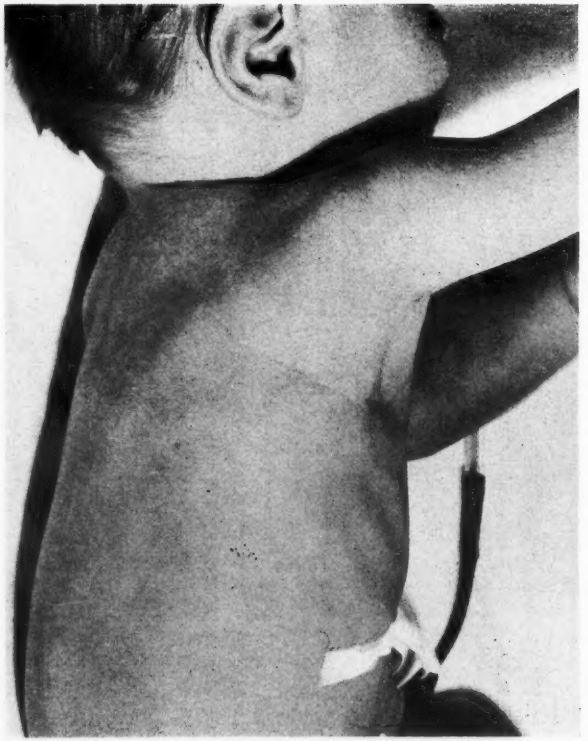

Fig. 7.—Patient at 185 days of age, showing site of incision for extrapleural ligation of esophagus.

and pharynx decreased somewhat. The catheter was removed from the extrapleural space after seven days (Fig. 5).

Following this stage, the baby's weight increased from 2,775 Gm. to 4,630 Gm. by March 27, 1940, when the third-stage operation was performed, bringing out the proximal blind pouch of the esophagus as a cervical esophagostomy. Postoperatively the temperature rose to 104.0° F. but rapidly became normal. The tip of the blind pouch was opened after two days and mucus drained freely from the esophagostomy, eliminating the necessity of aspirating the mucus from the mouth and pharynx.

Except for bilateral otitis media early in May, the baby has shown satisfactory progress and on May 22 weighed 5,105 Gm. (Figs. 6 and 7.)

COMMENT

By dividing the operative procedure into several stages, each of which has been well tolerated by the patient, the major problems presented in the management of these cases have been successfully handled. It has been demonstrated that extrapleural ligation of the fistulous communication of the esophagus to the trachea is readily accomplished.

While re-establishment of the continuity of the esophagus must be delayed in this case, the probability of survival of the patient is greater than if direct anastomosis of the upper and lower segments of the esophagus is attempted.

CONCLUSION

A method of surgical management of cases of congenital atresia of the esophagus with tracheoesophageal fistula is described. After a preliminary gastrostomy, an extrapleural ligation of the communication of the lower segment of the esophagus to the trachea is carried out. The proximal blind pouch of esophagus is exteriorized to make a cervical esophagostomy. The cervical esophagostomy and the gastrostomy can be connected by a rubber tube for feeding. Later, an antethoracic esophagoplasty can be done to re-establish continuity of the gastrointestinal tract.

A case is reported in which the described procedure was used. The patient was alive and well at 180 days of age.

AUTHOR'S NOTE: The infant is alive and well at 1 year and 7 days of age. His present weight is 8,190 Gm., and except for occasional upper respiratory infections, his development has been uneventful.

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PRIMARY BRONCHIOGENIC CARCINOMA WITH RUPTURE OF LEFT PULMONARY ARTERY

Case Report With Autopsy Findings

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THE case presented in this paper is that of a middle-aged man who died suddenly from a pulmonary hemorrhage. It is of interest because there was a history only of dyspnea and hemoptysis; the physical signs and roentgenograms gave no clue to the pathologic anatomy found at autopsy. We thought it worth while to present this case, as it shows the difficulties encountered at times in making a diagnosis of bronchiogenic carcinoma with its secondary effects; it also stresses the importance of early bronchoscopic examination.

History.—Mr. C. H., a 56-year-old white carpenter of good habits, entered the Presbyterian Sanatorium on Dec. 9, 1939, because of dyspnea and hemoptysis. Eleven months before, he had had a severe cold which kept him in bed five weeks. Although he returned to work and gained 10 pounds in weight, he experienced from then on a gradual but steady development of shortness of breath on exertion. The dyspnea grew rapidly worse in the six weeks before death, and hemoptysis of varying amounts occurred frequently during this period.

Physical Examination.—The patient was well developed and nourished, and weighed about 160 pounds. His chest was rather large, symmetrical, and the anteroposterior diameter was increased. The percussion note was slightly impaired over the left base, but there was fair resonance throughout the remainder of the chest. The breath sounds were bronchovesicular in character, and an expiratory wheeze with a few scattered rhonchi was noted above the midscapular lines. Blood pressure was 140/90, and pulse, 72; the heart was normal. The remainder of the physical examination was essentially negative.

Laboratory examination showed: red blood cells, 5,990,000; white blood cells, 13,650; hemoglobin, 83 per cent. The differential count revealed 65 per cent neutrophiles and 35 per cent lymphocytes, with marked toxic granulation of all the white blood cells. The blood Wassermann reaction was negative, and urinalysis was negative. No tubercle bacilli were found in the sputum.

Fluoroscopic examination in the left lateral position revealed that the cardiovertebral space at the level of the lower thoracic vertebrae could not be clearly visualized. Stereoscopic roentgenograms of the chest made on admission showed that the bronchial markings were much increased. A pleural effusion was noted over the left base, but the parenchyma of both lungs was clear.

On Dec. 14, 1939, the entire bronchial tree was injected with lipiodol. Fluoroscopy and posteroanterior and oblique roentgenograms revealed that the lower part

From the Maytag Laboratories of the Southwestern Presbyterian Sanatorium, Albuquerque, N. M.
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of the bronchial tree appeared normal on both sides and was well outlined by the lipiodol. Bronchoscopic examination was advised, but before this could be done, the patient died suddenly on Dec. 16, 1939, from a pulmonary hemorrhage.

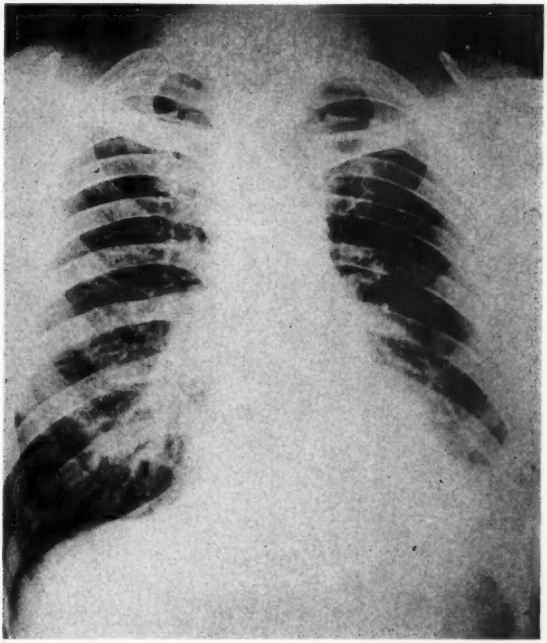

Fig. 1.—Posteroanterior film made on admission, Dec. 9, 1939.

Post-Mortem Examination.—After removal of the breast plate, the heart and lungs appeared normal in size, shape, and position. The right pleural cavity contained no fluid. The lung was entirely free on the right, and was crepitant throughout. The left pleural cavity contained from 300 to 400 c.c. of clear, straw-colored fluid. There were no adhesions on the left side. The left upper lobe was crepitant throughout and filled almost the entire hemithorax, the lower lobe being contracted to the size of a small grapefruit, red and glistening. A tumor mass, approximately 10 cm. in length and 3 cm. in diameter, involving only the contiguous lymphatics, was firmly adherent to the left dorsal spine and aorta. It was directly adjacent to the contracted lower lobe. All thoracic viscera were removed in one mass. Gross sections through the lungs at the level of the tracheal bifurcation and the eighth and ninth dorsal vertebrae showed a dense, gray, hilar infiltration, which penetrated the middle and posterior mediastinal structures. The left inferior bronchus was occluded. There was necrosis in the left hilum, with rupture into the left pulmonary artery, at about the level of the seventh dorsal spine. The left upper lobe was emphysematous, and the lower lobe was atelectatic. No tumor infiltrations were found in the pulmonary parenchyma. Although metastasis had occurred to the tracheobronchial glands, it was not found in any other part of the body.

Microscopic Sections.—The tumor growth showed dense, fibrous tissue containing many islands of malignant epithelial cells, characteristic of a bronchial adenocarcinoma.

DISCUSSION

Carcinoma arising in the large bronchi tends to form masses about the bronchus, usually at the root of the lung, with stenosis and ulceration of the lumen. In the case presented, the tumor occluded the left inferior stem bronchus, causing atelectasis of the left lower lobe and compensatory emphysema of the left upper lobe. Although these secondary effects were extensive, they did not interfere very much with respiratory function or bronchial drainage, and they did not produce significant abnormal physical signs.

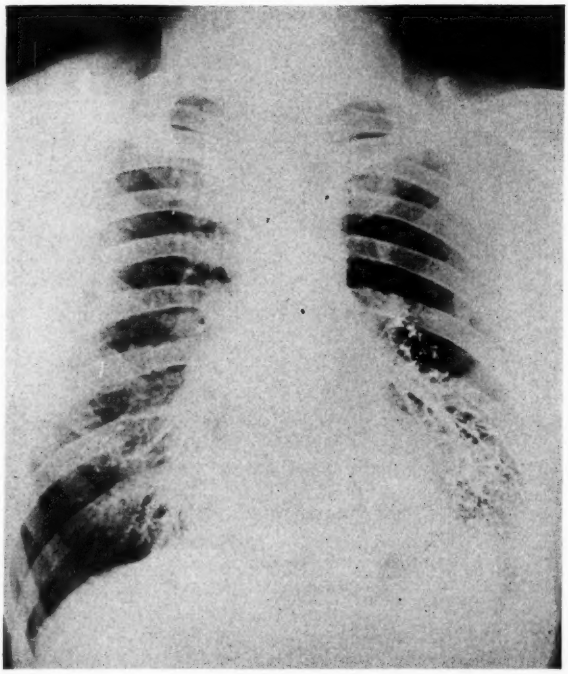

Fig. 2.—Posteroanterior film made following injection of lipiodol, Dec. 14, 1939.

Roentgenologic studies with and without iodized oil were of no aid in arriving at a diagnosis. In comparing the left lung at post-mortem examination with the x-rays, it was noted that, because of the obstruction of the inferior lobe bronchus, the lipiodol had not entered the lower lobe. The upper lobe almost completely filled the left hemithorax, so that the shadows, cast by the lipiodol in the superior lobe bronchus, were mistaken for the bronchial markings of the lower lobe.

Although the tumor was far advanced and beyond treatment, bronchoscopy was the only procedure by which a diagnosis could have been

made. By this method of examination, a diagnosis can be made earlier than by any other method and before secondary effects of bronchial occlusion are established. Therefore, when no roentgenologic changes of the chest are noted, bronchoscopy is indicated in all patients with a history of cough, dyspnea, and hemoptysis.

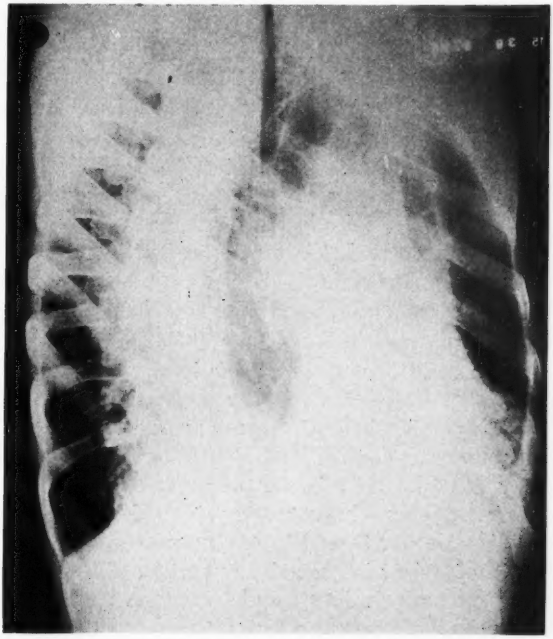

Fig. 3.—Oblique film made following injection of lipiodol, Dec. 14, 1939.

SUMMARY

A case of primary bronchiogenic carcinoma with rupture into the left pulmonary artery is reported. The difficulties in diagnosis and the autopsy findings are discussed. The importance of early bronchoscopic examination is stressed.

PRECISE LOCALIZATION OF PULMONARY ABSCESS

THE "SPOT" METHOD

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THE recent work of Neuhof and Touroff[1] has demonstrated the advisability of treating putrid lung abscess in the acute stage by a one-stage drainage operation. Because of the excellent results which they have reported in a large series of cases, this operation bids fair to become the treatment of choice in almost all cases of putrid abscess of the lung in the early stage, but if results comparable to those achieved by these authors are to be obtained by others, accurate preoperative localization of the lesion and its overlying adhesions is essential.

In the majority of cases of acute lung abscess, the peripheral situation of the lesion makes roentgenographic localization a comparatively simple matter. By means of the ordinary dorsoventral and lateral views, an abscess in the mesial half of the pulmonary field usually can be located with accuracy. The rib level at which the abscess is located is determined by counting the ribs on the roentgen film. The distance from the transverse process in posteriorly situated lesions, and from the costochondral junction in those situated anteriorly, may be measured. The localization of lesions in the axillary portion of the chest, which is a more difficult problem, is determined from films made in the oblique and lateral positions. From these views it may be determined whether the abscess is more superficial in the anterior or posterior axillary lines, or in the midlateral portion of the chest.* In the case of the latter, the rib levels may be determined from the lateral view. In the case of abscesses situated more anteriorly or more posteriorly, one oblique view shows the region in which the abscess is most superficial. The opposite oblique view is utilized for determining the rib level of the lesion.[2]

From these examinations, the situation of abscesses lying directly beneath the chest wall (approximately 95 per cent of all cases) may be determined. Indeed, experience has shown that, with the possible exception of abscesses in the midlateral portion of the lung, an accurate idea of the position of the abscess with relation to the chest wall may be obtained from roentgenologic examination in the dorsoventral, lateral, and oblique views.

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*The term midlateral is used to denote the area occupying the lateral portion of the chest between the anterolateral and posterolateral curves. It is used in preference to the common term axillary because, anatomically, the axilla corresponds to only the anterior two-thirds of this region and because the latter term is used by roentgenologists to denote the entire lateral portion of the pulmonary field.

In the case of abscesses in the midlateral portion of the lung, an error may be made because of the difficulty in identifying and counting the ribs in the lateral view and the difficulty in obtaining a precise lateral projection. It has been our experience, however, that errors may be made in approaching the abscess despite the accurate information at hand. The main difficulty lies in utilizing, at the time of operation, the information gained from the films as to the location of the abscess. It is not always possible to transfer the knowledge of the location of the abscess as gained from the roentgenogram to the actual operative field. The first and most obvious difficulty is that of identifying the rib which is exposed at operation. In stout persons, rib counting and identification are extremely difficult, and even in thinner patients it is often impossible to identify the ribs adjacent to the spine high in the interscapular region, in the upper axilla, or in the suprascapular region. Identification of the lower ribs by palpation depends upon the recognition of the twelfth rib and it may be so short as not to be palpable. Occasionally, an anomalous thirteenth rib, which leads to an error in rib identification, may be present. The upper ribs usually are counted by locating the prominent second sternochondral junction, but in the case of anomalies of the bony structures in this region, particularly in those not infrequent cases in which the first rib is situated lower than is usual, errors have been made. Thus, rib identification on the operation table often is impossible, and factors may be present that make identification erroneous even when the surgeon believes himself certain of its correctness.

In addition to the proper identification of the rib level at the operating table, the surgeon must know the distance between the abscess and some immovable landmark along the course of the rib. This distance may be determined from the dorsoventral films in the case of abscesses that are near the transverse process of the vertebra at one end, or the costochondral junction at the other. However, there is no satisfactory landmark from which the location of an abscess situated at a distance from these points, may be judged with accuracy. This is due to the fact that the curve of the ribs, as they approach the anterior or posterior axillary bends, foreshortens their roentgenologic projections. But, even if the distance could be determined, the surgeon would have no way of measuring it unless he stripped the ribs in order to measure directly the distance from one of the fixed bony points to the site of the abscess. Measurement on the skin surface leads to error because of the varied thickness of the chest wall, and there is no way of compensating for this error.

From the foregoing, it is obvious that rib counting and measurement are not dependable for locating abscesses on the operating table, although the lesions may be accurately localized on the roentgenogram.

A direct method of transferring the roentgen localization of the abscess to the operative field is required for purposes of direct entry into the abscess through the overlying adhesions. The most obvious method is to mark upon the skin, with indelible material, the spot underneath which the abscess lies. This can be done only at fluoroscopy, but unfortunately, it is often impossible to visualize the abscesses fluoroscopically in the required positions (oblique and lateral) when the lesion is located in the outer portion of the pulmonary field. In the case of abscesses located more mesially, this method is feasible, but it leads to error because the mark on the skin moves in relation to the underlying abscess when the patient's position is changed. Even if the mark is placed on the skin with the patient in the same position in which he is to be at operation, the surgeon is still left with no accurate guide once the incision has been made and the skin retracted. Despite all precautions taken to eliminate the factor of skin movement, this method, even combined with the greatest care in rib identification and measurement of distances, frequently has led us far enough away from the abscess to permit pleural entry or entail repeated puncturing of the lung in an attempt to locate the abscess.

After encountering all the difficulties mentioned above, it became evident that there was need for an immovable guide in the region of the abscess to be used by the surgeon in locating the lesion after exposing the ribs. Neuhof suggested the introduction of a needle into the intercostal space in the region of the abscess, not to be removed until the rib over the abscess was ready to be resected. However, this proved impractical because the needle slipped from its position during the manipulations incidental to the roentgenographic examination and the transportation of the patient to the operating table. A deeper insertion of the needle might lead to pleural contamination or to infection of the chest wall.

It therefore appeared necessary to devise a marker which would fulfill the following requirements:

1. It should be radio-opaque, so that the distance and direction between the abscess and the marker could be determined roentgenologically.
2. The relative position of the marker should remain unchanged with changes in the position of the patient.
3. The marker should be found easily by the surgeon.
4. It should lead to no harmful complications.

The method, which I term "spot localization," consists in placing a colored spot of radio-opaque material in the intercostal space in the approximate region of the abscess. The relationship of this spot to the abscess and the overlying rib is determined by appropriate roentgenologic examinations. The colored spot is located at the operation. Knowing the relationship of this fixed spot to the rib overlying the abscess, the proper rib is easily located. By measuring along this rib a distance

previously determined from the roentgen examination, the exact point is determined where the abscess is most superficial and where adhesions are expected to be present.

TECHNIQUE OF SPOT LOCALIZATION

Preliminary roentgen films in the erect dorsoventral, both obliques, and lateral positions are made, and the approximate location of the abscess is determined. In the case of more mesially situated lesions, one need know only the rib level and the approximate distance from the spine or costochondral junction. In those that are more laterally situated, it should be determined whether the abscess is situated in the midlateral portion of the chest, or whether it is located under the anterior or posterior bends of the ribs. The colored radio-opaque material is then injected into the intercostal muscle in the region of the abscess. If the lesion is visible at fluoroscopy in the proper position, a point directly over the abscess is selected for the injection during this procedure. If not, the injection is made at the approximate site of the abscess as determined from the roentgen films. The injection is carried out as follows:

During fluoroscopy, with the patient in the recumbent position, the finger is placed upon the chest over the rib directly above the abscess, and this point is marked on the skin with ink. The area is painted with iodine and the latter washed off with alcohol, precaution being taken that the patient remains in the same position throughout the entire procedure. The superficial skin is anesthetized by an injection of procaine. A deep insertion is now made until the rib is encountered by the tip of the needle. A small quantity of the procaine is injected into the periosteal tissues and into the intercostal space just above the rib. Two-tenths cubic centimeter each of a ½ per cent solution of aqueous methylene blue and of a 40 per cent solution of iodized oil, the latter previously warmed so that it flows freely, are drawn into a tuberculin syringe, utilizing a short beveled needle of 19 gauge, measuring 2½ inches long for thin patients and 3 inches for stouter ones. The needle is inserted at the level of the selected rib and its course is directed upward until the needle strikes the upper border of this rib. Even if the rib cannot be palpated because of the thickness of the overlying tissues, the position of the rib is known from the experience with the preceding procaine injection. When the impact of the needle against the rib is noted (this may be checked by repeated short strokes of the needle against the rib), the needle is tilted upward more sharply until it is felt to slide over the upper border of the rib into the intercostal space. Then the needle should be inserted more deeply for ⅛ inch. Now, it may be assumed that the needle has penetrated the deep fascia overlying the intercostal muscle, and the injection of the entire contents of the syringe is made into this muscle. The needle is left in situ, the syringe is disconnected, and

another syringe containing 0.2 c.c. of methylene blue is attached. The needle is withdrawn for a distance of ⅛ inch, and a very small quantity of the blue injected. The needle is then withdrawn ¼ inch at a time, and at each withdrawal small quantities of blue are again injected until the needle has been withdrawn from the chest.

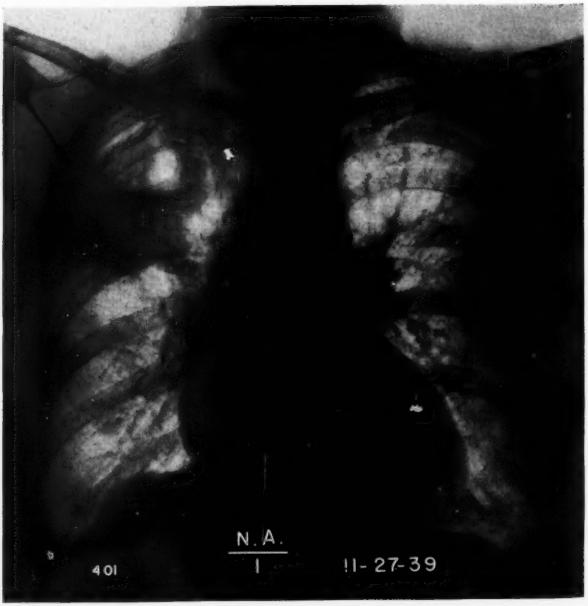

Fig. 1.—Case 1. Dorsoventral projection of large abscess situated in the axillary segment of the right upper lobe. The abscess is projected from the level of the first to the third ribs anteriorly, with the fluid line at the height of the second rib. Posteriorly, it is projected from the level of the third intercostal space to the seventh rib. The spot is situated in the fourth intercostal space and appears to be located over the upper portion of the abscess. In this case 0.4 c.c. of iodized oil were used. Note that the material has extended along the fibers of the intercostal muscles and has not crossed the ribs.

If the injection has been properly performed, there will be a spot of iodized oil mixed with methylene blue localized to the intercostal space, for the attachment of the deep fascia to the ribs will prevent the spread of the oil beyond this attachment. The oil and methylene blue spread for a variable distance along the intercostal space, but rarely for a distance of more than 1½ inches. The additional injected material, consisting solely of methylene blue, will be situated more superficially in the muscles, fascial planes, and subcutaneous tissues. It spreads for a variable distance, and is displaced from the main mixture of oil and blue, depending upon the subsequent movement of the tissues. However, the main mass containing the iodized oil will remain fixed, as stated above, despite the movement of the patient.

Fluoroscopy is now repeated in order to determine that a proper injection has been made. This may be assumed to be the case if the radio-opaque material is seen to lie within the intercostal space in the form of a streak between the ribs, and to remain immobile in relation to the ribs on movement of the patient and on respiration. If the spot has not placed within the intercostal muscle, it is not too late to repeat the procedure, for the properly placed spot can be recognized both on the roentgen films and in the operative field.

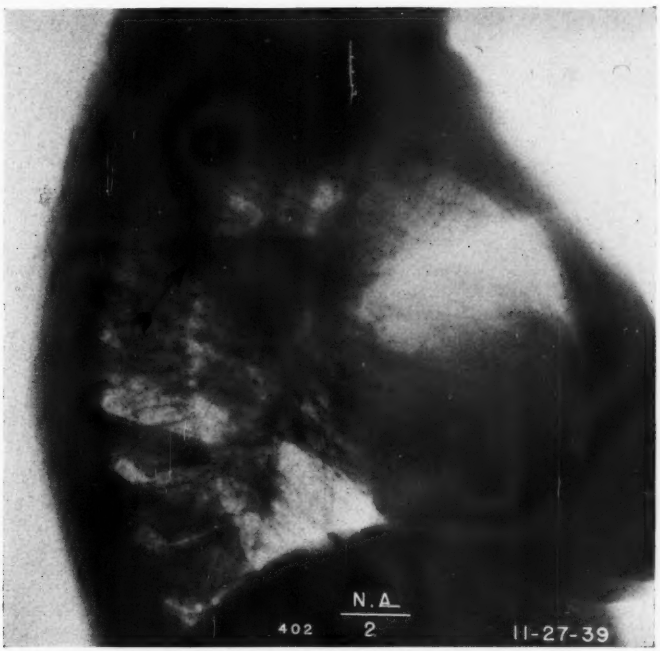

Fig. 2.—Case 1. Lateral view. This is the view which shows the least distortion of the ribs directly over the abscess, and is the one used for its localization. Although the ribs cannot be identified on the roentgen film, it is clearly demonstrated that the rib to be resected is the segment directly above the anterior portion of the spot and that the center of the abscess is situated directly above this at the level of the third intercostal space.

Roentgen films are made again in the positions previously mentioned: namely, dorsoventral, lateral, and both oblique positions. As in the preliminary examination, the patient should be in the erect position. If the patient is very ill, a supporting binder may be required for this purpose.

INTERPRETATION OF LOCALIZATION WITH REFERENCE TO THE SPOT

The location of the abscess in relation to the spot is judged from that film in which there is the least distortion of the length of the rib overlying the abscess. In abscesses lying in the mesial half of the

pulmonary field, the dorsoventral film (or the ventrodorsal if one prefers to use this projection) shows the least foreshortening and will therefore indicate the distance relationship between the spot and the abscess most accurately. When the abscess is situated in the midlateral portion of the chest, the lateral projection will cause the least distortion of the distance between the spot and the abscess, and it is from this film that the distance between the abscess and the injected spot will be

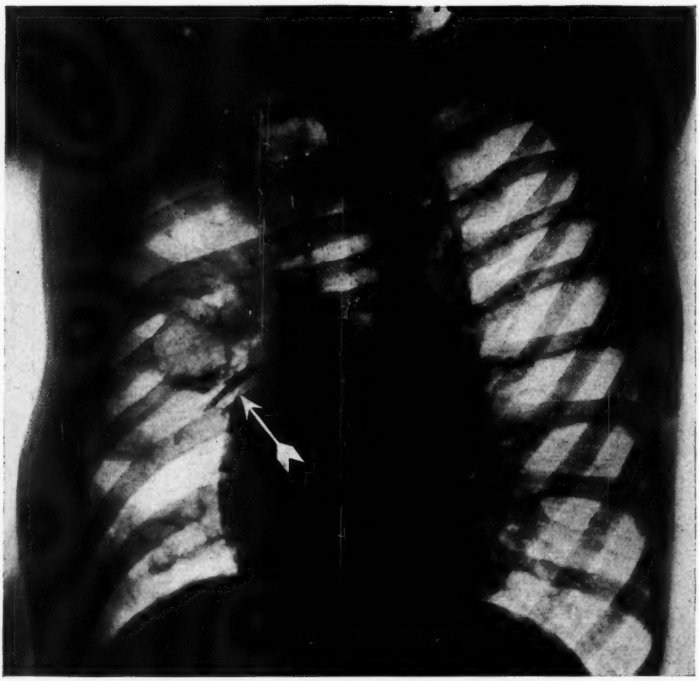

Fig. 3.—Case 2. Cavity in lower posterior part of axillary segment of right upper lobe. The position of least distortion is the left oblique. The lesion extends from the lower border of the fourth rib to the sixth intercostal space. It is situated directly above the anterior end of the spot, which is in the sixth space. Iodized oil, 0.2 c.c., was used. A spot of this size is preferable to that used in Case 1.

judged. In the case of abscesses situated beneath the bends of the ribs in either the antero- or posterolateral regions, the oblique projection of the chest on a film placed tangential to the site of the lesion is the one used for localization. Thus, given an abscess situated beneath the bend of the rib in the anterolateral region on the right side or in the posterolateral region on the left side, the right oblique position (right shoulder to the film) is the one that is used. On the other hand, the left oblique view is used in the case of lesions situated in the anterolateral region on the left side and in the posterolateral region on the right.

On examination of the film selected as showing the least distortion, a streak is seen about ½ inch in width and 1 inch in length, confined to the intercostal space at the site of the injection. This usually shows striations caused by the muscle fibers between which the iodized oil has penetrated. The relationship between the center of the abscess and the streak is determined in order to select the proper portion of rib to be resected in drainage of the abscess. The vertical distance between the abscess and the spot is noted according to the number of ribs that intervene, and the horizontal distance is noted by measurement of the actual distance along the rib between the spot and the abscess. It it not necessary to identify the number of the rib, although this is easily done on the roentgen film.

The operative incision is guided by the chest puncture mark which has been stained blue by the injection, and the incision is further guided by the blue-stained tract that was made as the needle was withdrawn, until the intercostal space stained by blue is encountered. This is marked by a blue spot bounded sharply by the ribs above and below it. The lateral borders of the spot are also sharply demarcated. The small portion of rib to be resected is selected in accordance with the relationships that have been determined, using this spot as a guide after the superficial tissues have been dissected free from the intercostal muscle and the adjacent ribs. If the injection has been accurately placed in relation to the abscess, a rib adjacent to the injected intercostal space will be the one resected. In occasional cases where the injection has been made at some distance from the abscess, a rib one or two spaces distant from the spot may be the proper site for resection. If the spot has been properly made and the films studied in the proper projection, there is no difficulty in locating the abscess by superficial aspiration after resection of a small portion of rib.

Utilizing the "spot" method for the localization of lung abscess in the last twenty-two cases which have been operated upon in our institution, we have encountered the following difficulties:

1. Difficulty in stout patients in shifting the needle from the rib to the intercostal space above it.
2. Injection of the material too superficially.
3. Injection of the material too deeply.
4. Difficulty in locating the intercostal spot because of its distance from the superficial puncture mark.
5. Injection of the material at a distance from the abscess.

The solution of these difficulties lies in the following points of technique:

1. Directing the needle upward to reach the upper border of the rib with the injecting needle before plunging the needle still further upward into the intercostal muscle. This permits the needle to be

shifted easily without loss of the sensation of the position of the end of the needle. A needle long enough for the purpose must be used. It should be remembered that the needle should not be inserted to its hilt if breakage and loss of the needle are to be avoided.

2. No attempt should be made to inject material into the intercostal space without first feeling the impact of the needle against the rib. This serves for orientation that will prevent too superficial or too deep an injection.

3. A superficial injection is recognized at fluoroscopy by displacing the soft parts by manipulation or movement of the patient's arm. If the spot is seen to move, it has been placed too superficially.

4. In one case the injection was made too deeply and the blue was not found until after the rib had been resected. The fact that the spot has been made too deeply can be recognized on the films made in a plane perpendicular to the tangent of the lung abscess; i.e., in a plane perpendicular to the film used in measuring the distance between the lung abscess and the spot. In this film the depth of the spot in relationship to the ribs and chest wall can be determined. The injection may be repeated or the rib resection may be carried out in accordance with the ordinary principles of roentgen localization, and the blue spot located on the deep side of the rib before the lung abscess is aspirated.

5. The additional injection of methylene blue without iodized oil in the more superficial part of the needle tract serves as a guide in finding the intercostal spot at operation in those cases in which the tissues have shifted in moving the patient. The shift between the puncture mark and the intercostal spot will be minimized if the spot is injected with the patient in approximately the same position as that in which he is to be operated upon. In any event, rib resection should not be carried out until the intercostal spot has been found, even if this requires rather extensive dissection for its disclosure. It is easier to find the spot of blue in the intercostal tissue than it is to find an abscess in the lung by blind puncture.

It is obvious that some experience with the above-described method is required for its proper use in the localization of lung abscess. Moreover, it necessitates the making of additional roentgen films. Nevertheless, the importance of precise localization of the abscess is so great, and the method so useful in eliminating errors incidental to rib counting and measurements, that it should be used as a preliminary step before drainage of a lung abscess is attempted.

SUMMARY

The difficulties in localization of lung abscess are discussed.

The main source of error in locating an abscess of the lung has been incurred in transferring the roentgen image of the location of the abscess to the thoracic cage as it is seen in the operative field.

This source of error is obviated by establishing a fixed spot in the intercostal muscle recognizable on the roentgen film and at the operating table.

The material selected for the injection is a mixture of 40 per cent iodized oil and 0.5 per cent methylene blue.

The technique of the injection is described.

The method of localization of the abscess in relation to this spot is discussed.

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PLEUROESOPHAGEAL FISTULA IN EMPYEMA

Report of a Case

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Columbus, Ohio

PLEUROESOPHAGEAL fistula in empyema is rare enough to warrant a case report, especially since the cause of the fistula is very unusual, as was also the hospital course of the disease.

Perforation of the esophagus by foreign bodies, corrosive poison, and carcinoma is not common. Perforation of the esophagus by empyema is almost unknown. I have failed to find a case reported in the literature wherein a pleuroesophageal fistula was produced by a drainage tube in an empyema cavity.

Blauvelt[1] reviewed eight cases of pleuroesophageal fistula due to empyema which were collected from the literature and one additional case of his own. He also reviewed two cases due to intrathoracic suppuration of unknown origin. Phillips[2] reported a case due to empyema, and Wallfield[3] reported a case of perforation of the esophagus followed by empyema and pleuroesophageal fistula. All cases reported have occurred since Arbuthonot Lane advocated primary rib resection for acute empyema in 1882. The first case reported was that of Voelcker in 1891.

Blauvelt suggests that pleuroesophageal fistula may be produced in four ways: (1) Perforation of the esophagus in the wall of an empyema by mediastinal lymph nodes. No cases reported, but theoretically possible. (2) Perforation of a diverticulum of the esophagus. No cases reported, but theoretically possible. (3) Perforation of an esophageal peptic ulcer producing an empyema. One case reported. (4) Perforation of a normal esophagus by an empyema. The majority of cases probably fall into this group.

The patients in his series were treated by: (1) thoracotomy by rib resection, (2) feeding through esophageal tube, (3) gastrostomy, and (4) direct closure of fistula. The mortality rate in the eleven cases reported by Blauvelt was 44 per cent.

CASE REPORT

A 7-year-old white girl was admitted to the Children's Hospital on Jan. 27, 1939. She had developed lobar pneumonia in April, 1938, and was ill with pneumonia for a period of five weeks. Following this, a right-sided empyema developed.

On May 12, 1938, she was admitted to a near-by hospital. Her temperature on admission was 105.4° F. The pulse rate was approximately 200; the respiratory

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rate, approximately 114. Physical examination, according to the hospital records, showed the interspaces to be bulging and the right chest to be solid to percussion. The heart was pushed to the left.

On May 14, 1938, the chest was aspirated, and approximately one pint of pus was removed. A section of the ninth rib was then removed posteriorly, and the empyema cavity was drained by a heavy, hard rubber tube.

The patient continued to improve, but on May 31, 1938, cocoa came through on the dressings. Lipiodol and barium injections of the esophagus during fluoroscopy revealed no fistula between the esophagus and the empyema cavity. However, the patient was then given methylene blue by mouth, and the methylene blue came through on the dressings, thus demonstrating a pleuroesophageal fistula.

The fistula apparently closed spontaneously on June 3, 1938. However, on July 16 it again opened, and food and methylene blue came through on the dressings.

It apparently again closed spontaneously on July 22. On September 17 the tube in the empyema cavity was removed, and the wound subsequently healed.

The temperature, however, rose. On Oct. 18, 1938, the wound was reopened; two empyema pockets were drained; and the hard rubber tube was again placed in the empyema cavity. Biopsy of the wound margins and of the pleura showed granulation tissue but no evidence of tuberculosis.

On Jan. 24, 1939, the patient suffered a profuse hemorrhage from the thoracic wound, and on Jan. 26, 1939, she again had a hemorrhage.

She was admitted to Children's Hospital following the second hemorrhage, at which time the pleuroesophageal fistula was still open. On admission, the hemoglobin was 93 per cent; R.B.C., 4,780,000; W.B.C., 6,550. The tuberculin test was negative. Differential count: polymorphonuclear leucocytes, 48; small lymphocytes, 52 per cent. Wassermann and Kahn tests were negative. On Jan. 30, 1939, a culture was planted on Petroff's media and was negative for tuberculosis. Smears of the wound were also negative for tubercle bacilli. The tuberculin test was negative.

The drainage tube was of hard rubber and was approximately 9 inches long and ⅝ inch in diameter. The tube entered the right thorax through the defect in the ninth rib in the scapular line about 1½ inches below the inferior angle of the scapula. The tube was directed medially and ran parallel to the ninth rib. The tube was removed, and the intrathoracic portion of the tube was between 3 and 3½ inches in length. When laid upon the chest wall in apparently the same position it occupied while within the thorax, the tip of the tube reached to the mid-spinal line.

X-ray examination showed some evidence of slightly thickened pleura at the site of the drainage tube, but no empyema or effusion was seen. The pleuroesophageal fistula closed spontaneously. Esophagoscopy was done Feb. 15, 1939, under ether anesthesia with a 6 mm. esophagoscope, but no evidence of fistula was seen.

The patient made an uneventful recovery. There has been no return of the empyema or of the pleuroesophageal fistula.

DISCUSSION

Pleuroesophageal fistula, due to trauma produced by a foreign body within the esophagus, causing perforation of the esophagus and subsequent empyema, is not uncommon. Perforation of the esophagus by the erosion of an empyema is quite rare. Perforation of the esophagus by a hard rubber drainage tube introduced into the empyema cavity with resulting erosion of the esophagus is extremely rare, and, to the best of my knowledge, this is the only case of its kind.

This case should serve to condemn the use of hard rubber tubing in the drainage of an empyema cavity. Surgeons are generally familiar with the erosion of the lung produced by hard rubber tubing with subsequent bronchopleural fistula and are also familiar with the erosion of intestinal wall by hard rubber tubing and subsequent fecal fistula. It is important to note that, following the removal of the hard rubber tube, the fistula and the track closed spontaneously.

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WHEN IS OLEOTHORAX INDICATED?

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TO MATSON[1] and MacKenzie[2] is due the credit of introducing the use of paraffin oil in the treatment of empyema. Bernou,[3] in 1922, described its use in compression therapy, and since that time there has developed a widespread use of various oils in collapse therapy, either intrapleurally in pre-existing artificial pneumothorax or as a tampon in extrapleural pneumothorax. A large amount of literature on oleothorax has appeared since 1922, and there seems to be a fair agreement that this method of compression is a valuable addition to our present methods of treatment.

Joannides[4] later added materially to our knowledge of the various oils and their particular uses in medicine and trade and repeated the main indications for oleothorax. Briefly stated, the indications for oleothorax may include: a preceding pneumothorax; an effusion with or without pneumothorax; a rapid absorption of air leading to an obliteration of the pneumothorax collapse; the prevention of adhesions; an antiseptic collapse with medicated oils; the fixing of a mobile mediastinum, particularly in mediastinal hernia; and the collapse of rigid wall cavities.

Improved methods in collapse therapy, extrapleural thoracoplasty, and, to a degree, extrapleural pneumothorax, have very materially changed our viewpoint about the use of oleothorax. Today such procedures are attended with a low mortality and with more permanent results.

We believe that a review of results will lead us to the surgical approach whenever feasible and possible. It often happens that oleothorax becomes only an intermediary stage, and we feel that usually it is detrimental to the final result.

Let us first consider the various indications as given above.

Pleural Effusion.—We do not know how frequently artificial pneumothorax, following removal of fluid, is used. To us it would appear that a definite distinction should be made between acute pleurisy with effusion, as the result of a minimal subpleural lesion, and that occurring with a reasonably extensive parenchymal involvement, with or without persistent pleuritis and effusion.

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There seems to be no definite rationale for converting the former into a hydropneumothorax and certainly not an oleothorax. Experience teaches us that adequate rest treatment stabilizes the pulmonary condition, and records of subsequent breakdowns, some years after such an experience, do not appear outstanding in our histories. Actually many patients made permanent recoveries with what we felt was inadequate treatment.

If, on the other hand, an extensive lesion is underlying the effusion, then consideration should be given to the treatment of the patient with artificial pneumothorax. If this proves successful and an adequate compression can be maintained, with a disappearance of the fluid, then at least that phase of treatment is behind us. If, however, an obliterative pleuritis occurs, we have then to consider oleothorax. We still have existent an extensive pulmonary disease, partially controlled. Of what benefit then can an intermediary stage of oleothorax be to our final treatment? We shall probably be left with a pocket of oil and exudate that prevents the ultimate success of an extrapleural thoracoplasty which we felt from the beginning was the most intelligent final attack.

Rapid Absorption of Air, Leading to an Obliteration of the Pneumothorax.—This difficult complication is given as an indication for oleothorax. Although our own experience with this procedure is very limited, we feel that, even if more frequent refills of air are required, the amount of compression that can be secured is sufficient to aid in the stabilization of the disease. In time such treatment, along with bed rest, often creates sufficient pleuritis to make the pneumothorax effective. Furthermore, we have not created an extensive pleuritis that is often associated with oil.

It would be well to consider the work of Ellwart, Saley, and Willis,[5] who experimented with intrapleural injections of vegetable oils in rabbits. They found that vegetable oils may produce extensive grades of fibrinous and fibrous pleurisy with marked visceral distortion and displacement. This follows closely our own experiences in man. As our case reports show, in those patients who eventually had extrapleural thoracoplasty the pleura was markedly thickened and rigid in every instance, and in three of these, upon opening the pleura, the wall had a thick coating of plastic debris, made up of oil and exudate associated with an infiltration of the wall by crystalline masses, interposing a serious obstacle to ultimate collapse.

Obliterative Pleuritis.—It would appear that oleothorax would be of definite value in maintaining a tampon when the process was not too far advanced. There is, however, definite danger in this procedure. In one of our patients, C. A., oil was introduced in the hemithorax. A mild obliterative pleuritis was present before oleothorax was started. This condition was not improved, and eventually an empyema and bronchial fistula followed, undoubtedly aggravated by the oil.

This experience taught us that such patients must be under as constant observation as with artificial pneumothorax, as complications may occur a long period after the initial filling.

Antiseptic Collapse With Medicated Oils.—We believe this indication for oleothorax is a long existent artificial pneumothorax, without empyema. In other words, it is a preventive procedure.

It is assumed, therefore, that it is proved that oleothorax with gomenol, or some other antiseptic, will prevent the ultimate formation of hydrothorax and empyema. Are we at all certain that such measures either prevent or cure an empyema? All of us have passed through the gamut of various antiseptic agents in the thorax, and we wonder how effective any of them have been in uncomplicated tuberculous empyema. Are we misinterpreting natural recovery, uninfluenced by antiseptics? It still seems that the best approach to tuberculous empyema is by extrapleural thoracoplasty, attempting to create apposition of the pleural surfaces. Certainly oil in our hands has been a serious deterrent to this end.

Fixing of a Mobile Mediastinum, Particularly in Mediastinal Hernia.—The acceptance of this as an indication for oleothorax acknowledges the fact that oil is an irritant in the pleural cavity. It seems quite logical that oleothorax would produce a stiffening of the mediastinum. Even in this difficult complication, a successful pneumothorax is eventually possible. We at least have not introduced an irritant foreign body in the hemithorax.

Collapse of a Rigid Walled Cavity.—The closing of rigid wall cavities is always a difficult problem, and even the most extensive removal of ribs leaves us with 20 per cent of them unclosed.

With a rigid mediastinum and a compressible lung, oleothorax should accomplish a high degree of compression.

Today patients who present a chronic condition with heavy walled cavities should be considered as surgical prospects. Even acknowledging the temporary benefits of oleothorax, still we are usually presented with a fairly extensive upper or mid-lobe involvement of such an extent that their future is always jeopardized, even with artificial pneumothorax. It is this type of patient that eventually receives the greatest benefit from an extrapleural thoracoplasty, with the least operative risk, and it does not seem a sound procedure to delay the ultimate treatment by the use of oleothorax, and later find ourselves with a far more difficult task. As a result of our own bad results from such treatment, we have decided never again to disturb the status quo of the pleural condition by the use of oleothorax.

We now wish to report the histories of four patients, and give a viewpoint of our failures with oleothorax. We admit the possibility of the improper use of oil. However, in each instance the indication for extrapleural thoracoplasty was clear cut, even before oil was introduced in

the hemithorax. We followed the path of temporizing because we wished to avoid a surgical attack. The first patient, C. A., will be discussed in detail, as a type case. Only the highlights of the other three cases will be reported.

CASE REPORTS

CASE 1.—A 25-year-old man was admitted to the Saint John Tuberculosis Hospital on Sept. 12, 1928, and discharged on June 2, 1931. His symptoms were marked loss of weight, cough, sputum, hemoptysis, shortness of breath, fever, and rapid pulse.

In the spring of 1926, while in his first year at college, he developed an acute illness characterized by high fever, rapid pulse, and marked diarrhea, the last symptom persisting for some months. The physician suggested intestinal tuberculosis, but no further examination was made.

After graduation he went to work on one of the West India Islands, as an accountant, and shortly after his arrival he contracted malaria. Cough and sputum were associated with the onset of malaria. He continued working until June, 1928, when his symptoms of cough and sputum became worse. He was then discovered to have pulmonary tuberculosis. A short rest period relieved his symptoms. He was then ordered to return to Canada.

Examination at the clinic revealed a very sick man with an extensive and active disease in the left hemithorax, and a florid disease on the right, localized between the third and fifth ribs, anteriorly, and the fifth and seventh vertebrae, posteriorly. Sputum was positive for tubercle bacilli; urine, normal; temperature, 99° to 101° F.; and pulse, 80 to 100.

A roentgenogram showed a well-defined and rather dense area in the left lung, with evident cavitation well delineated by the interlobar fissure, between the upper and lower lobes. On the right side, a well-defined interlobar line was seen with scattered, fluffy areas throughout the entire side.

The final diagnosis was far advanced pulmonary tuberculosis.

The patient was desperately ill for a long period of time and was kept at rest. His pulmonary disease gradually subsided and was associated with marked clearing of the congestion on the right side. On May 14, 1929, artificial pneumothorax was instituted on the left side, with adequate collapse. All symptoms diminished, and sputum was rapidly converted. This state continued for several months when the pleuritis began to diminish the air pocket and there was definite re-expansion of the lung.

This was followed by an interesting sequence of events. At approximately three-month intervals the patient would become violently ill, with chills, fever to 104° F., pulse elevation to 120, nausea, and vomiting. This would continue for three to four days and would be followed by cough and expectoration. In all, he would expectorate some 3 to 4 ounces per day. This phase would gradually subside; the general and local symptoms would disappear. Sputum was always positive. At no time did an effusion occur in the pneumothorax pocket.

If oleothorax had been used as freely as it was later, evidently we would have carried out this procedure on the basis of results reported in obliterative pleuritis. However, it was not until a later admission, April 4, 1934, after a period of active work that this was considered. In the meantime the patient was able to work but persisted in his cyclic drainage, mentioned above.

On the second admission, a trial of 2.5 c.c. of gomonoleo, intrapleurally, was made. A marked febrile reaction, lasting one day, was the result, and eventually 500 c.c. of cottonseed oil were introduced. The patient received refills of 20 to 30

c.c. every two to three months. His sputum became negative, and the cyclic reactions disappeared; obviously the cavity had been closed. He was discharged on Aug. 9, 1934.

After leaving the hospital, he hitch-hiked to California, was not examined at any time, was out of work, and at one time was without food for days. He was finally deported to Canada, and arrived at this hospital on June 14, 1935. He then had a bronchopleural fistula and empyema. His sputum was still negative.

On June 25, 1935, 215 c.c. of material were aspirated—oil and tomato-colored pus, undoubtedly hemorrhagic, and without odor. Guinea pig inoculation revealed tubercle bacilli.

The condition was considered bad, and immediately thoracoplasty, following aspiration, was decided upon.

From June 27, 1935, to Sept. 1, 1935, four stages of thoracoplasty were completed, both posterior and anterior, with a total removal of approximately 80 inches of rib. Despite this extensive operative procedure, a hard-walled, slitlike pocket remained, requiring frequent drainage and instillation of Dakin's solution. In the meantime the bronchial fistula had closed.

In July the thoracic pocket was opened; a complete unroofing and saucerization of the empyema pocket was carried out; and the area was packed. Death occurred on July 30, 1936, from tuberculous pneumonia in the contralateral lung.

A review of this case, with the knowledge we now have, shows that it should have been evident that oleothorax was not indicated, even if it did close the tuberculous cavity.

An extrapleural thoracoplasty should have been the treatment of choice at the time the cavity remained patent. We then had only a mild pleuritis with which to contend. Ultimately we had a severe pyo-oleothorax and bronchial fistula, one of the most difficult complications presented to a surgeon.

CASE 2.—A graduate nurse, aged 23 years, had onset of tuberculosis (pleurisy with effusion) in December, 1928. She was treated at home and in sanatoria for a moderately advanced tuberculosis. Sputum was persistently negative. Recovery was slow. Then in June, 1931, she had an exacerbation.

Artificial pneumothorax started in October, 1931, and continued until March, 1932. Collapse was selective; pleura, adherent over the lower lobe; sputum, converted. Feeling that pneumothorax would eventually be lost, we introduced cottonseed oil into the pneumothorax pocket. The pocket proved to be very small, and within three months the oil was withdrawn.

Another exacerbation of tuberculosis occurred in October, 1936. Extrapleural thoracoplasty was decided upon, but operation was delayed until July, 1937, because of unstable disease.

Thoracoplasty, three stages, with incision of pleura during first stage, was undertaken from July 9, 1937, to Oct. 19, 1937.

The intrapleural pocket, even though oil had been removed three months after its introduction, revealed a markedly thickened wall with sheets of crystalline material embedded in the pleura. The pocket was filled with puttylike material mixed with pus and oil. On Nov. 19, 1937, the entire pocket was excised as completely as possible. Drainage continued for one year and three months.

Since pneumothorax was not satisfactory, thoracoplasty should have been the treatment of choice. The patient's condition was good, and she had already a known history of chronic tuberculosis with one exacerbation. The oil served no purpose but acted as a severe irritant, making the operative procedure complicated and dangerous.

CASE 3.—A man, aged 23 years, had a positive family history of tuberculosis. The first symptoms occurred in the summer of 1929. He entered the hospital on July 9, 1930. He had upper left lobe involvement with a cavity 4 cm. in size. Artificial pneumothorax effectively closed the cavity and resulted in good quiescence by June 18, 1932. He returned to the seminary. It was decided at another clinic to give oleothorax because of mildly progressive pleuritis, and it was felt that pneumothorax would be more difficult to maintain in an isolated missionary field.

In 1939, although he had never had hydrothorax, he developed this condition, followed immediately by pyothorax. Frequent aspirations and the use of an antiseptic, mercurochrome, did not influence the condition. Extrapleural thoracoplasty was decided upon, but it was then discovered that the condition in the contralateral lung was unsatisfactory. This necessitated the use of pneumothorax. At present he is having aspiration about every three weeks, and the artificial pneumothorax on the contralateral lung is being continued. The patient is nontoxic and eventually will have more surgical treatment.

If the pneumothorax were satisfactory, why introduce oil into the hemithorax? It would have been far better to have eventually lost the pneumothorax and allowed the pleura to become adherent. Extrapleural thoracoplasty would then have been the treatment of choice. Our problem is now complicated by an empyema, with difficulty in closure.

CASE 4.—This patient was a woman, aged 23 years. Onset of tuberculosis occurred during the winter of 1934. She entered the sanatorium in the spring of 1934 and was found to have extensive disease with cavitation in left upper lobe. Artificial pneumothorax was instituted. In November, 1934, the patient developed hydropneumothorax, requiring several aspirations. In June, 1935, after the fluid had disappeared, she had a small localized disease in the left upper lobe, with minimal symptoms. Pneumothorax and rest were continued, with quiescence of the new area on the right side. In September, 1936, at another institution, 1,000 c.c. of cottonseed oil were introduced, beginning with 2.5 c.c. gomenol, and continuing treatment with increasing amounts of cottonseed oil. The sputum remained consistently negative. Thoracoplasty was not deemed advisable on her first admission in September, 1937, because of the acute exacerbation of the contralateral lung. As much oil as possible was aspirated. She returned for reconsideration of thoracoplasty on June 7, 1939. Again oil was aspirated, and it was found that the hemithorax was filled with purulent fluid, tinged pink with mercurochrome. Tubercle bacilli were present on direct smear.

A four-stage extrapleural thoracoplasty (three anterior stages and one anterolateral stage) was completed. The wall was rigid and was compressed with difficulty. Even extensive rib resection left an empyema pocket with a capacity of nearly 100 c.c., necessitating drainage and a partial decortication. The patient is now left with an open drainage, but is feeling fine.

The procedure that we should have adopted was an extrapleural thoracoplasty when the artificial pneumothorax was not entirely successful, and before the formation of the empyema. There was no indication for oil at any stage of treatment, and we have only prolonged her treatment with less satisfactory results.

CONCLUSION

Our own experience with the use of oleothorax has been most unsatisfactory, and we feel that a different approach was indicated. Extrapleural thoracoplasty was indicated particularly in those patients with moderate to extreme unilateral pulmonary tuberculosis, progressive and adhesive pleuritis, and thick-walled cavities.

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THORACOPLASTY IN THE TUBERCULOUS DIABETIC PATIENT

A Clinical Study

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SURGERY in the diabetic patient has been the subject of many presentations. Chest surgery, especially thoracoplasty, in pulmonary tuberculosis associated with diabetes mellitus has been, however, only lightly touched upon in a few scattered instances. Izzo and his associates[1] have reported the performance of thoracoplasty in a 50-year-old woman with unilateral tuberculosis and moderately severe diabetes. The good result was attributed largely to careful preoperative preparation. Amorim and Castello Branco[2] in Brazil reported a single case. King[3] made casual reference to thoracoplasty in the diabetic patient, and Root[4] stated (without details) that several of his patients with diabetes and tuberculosis have been subjected to pneumothorax and thoracoplasty.

The relative frequency of occurrence and changed outlook in associated tuberculosis and diabetes have been dealt with in their varying aspects by McKean and Myers.[5-7] The point has been stressed by these authors that, while there are instances to the contrary, changes for better or worse in one disease usually reflect themselves in a parallel fashion in the other. A greater understanding of diet and insulin therapy directed toward the diabetes has worked, we believe, to the definite benefit of the pulmonary lesion as well. It would be expected, therefore, that measures effecting a better control of the tuberculosis lesion would lend themselves to the improvement of *both* the metabolic status and the infectious status.

For many years artificial pneumothorax and phrenic nerve surgery have been used unhesitatingly in the patient with diabetes and tuberculosis. The employment of thoracoplasty, on the other hand, in this group of patients was approached by both surgeon and medical man with decided trepidation. The long existent fear of major surgery in the diabetic patient uncomplicated by tuberculosis was partly responsible for this attitude. As a result, many patients with tuberculosis and associated diabetes were denied this form of chest surgery for some time.

It is our purpose, therefore, to review the experience in the Tuberculosis Division of Herman Kiefer Hospital with the group of patients

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having pulmonary tuberculosis complicated by varying degrees of diabetes mellitus on whom extrapleural thoracoplasty was performed.* The first patient in this group was operated upon in 1933 and is still living. From 1933 to 1939 a total of eleven patients have received this type of collapse therapy. Because some of these patients have been operated upon recently, we believe that any definite conclusion would not be warranted but that the data already obtained may be of interest.

Classification of Disease.—The classification of the tuberculous process is that adopted by the National Tuberculosis Association. Five patients had moderately advanced tuberculosis, and six, far advanced. Of these, one had intractable empyema. The diabetes is classified according to the insulin requirements immediately before operation: mild (cases requiring less than 20 units of insulin), moderately severe (requiring 21 to 40 units), and severe (requiring over 41 units). On this basis, four were mild; five, moderately severe; and two, severe.

Indications for Thoracic Surgery.—Thoracoplasty is considered to be indicated, as in the tuberculous patient without diabetes, when other collapse procedures failed to close cavities or effect a negative sputum, and when the general condition of the patient and an adequate amount of ventilating lung tissue warranted it. All but one of this group had cavities which were inadequately influenced by artificial pneumothorax or phrenic nerve surgery. The only patient without cavity had far advanced tuberculosis and empyema of proved tuberculous origin. Six of the eleven patients had sputum positive for tubercle bacilli prior to operation.

Surgical Preparation.—In the routine preoperative study of patients about to have thoracoplasty, the following tests are made: phenolsulphonephthalein excretion, blood nonprotein nitrogen determination, bleeding and clotting time estimation, and electrocardiogram. For the past two years it has been routine procedure to do a bronchoscopic examination on all candidates for thoracoplasty. Preoperative medication consisted only of morphine sulphate, ⅙ gr. Nitrous oxide-oxygen anesthesia was used in all instances.

Diabetic Management in the Operative Period.—The patients were controlled as well as possible in the presence of the infectious complication before operation. The criteria for control were (1) urine free of sugar, (2) blood sugar range between 80 and 150 mg. per 100 c.c. in the fasting state, and preferably throughout the day, (3) maintenance of weight as close to the standard normal as possible, and (4) the avoidance of hypoglycemic reactions.

The decision in favor of operative intervention was made in joint medical-surgical conference. The time for operation was recommended by the metabolic division and was based on satisfactory diabetic control. On the morning of operation, the usual "available glucose"† taken for breakfast was given as 10 per cent dextrose in normal saline by *slow* intravenous injection. After it was certain that the intravenous solution was being well taken, the usual dose of insulin was

*All of the operations were performed at Herman Kiefer Hospital by Dr. E. J. O'Brien and his staff except one case managed privately at Harper Hospital by this staff and one of us (G. C. T.).

†"Available glucose" is considered the total glucose in the dietary prescription representing 100 per cent carbohydrate, 58 per cent protein, and 10 per cent fat.

administered subcutaneously. On return from the operating room, more dextrose in saline solution was given, the total at this time being equivalent to the available glucose usually received in the noon meal. Insulin was given as above. Alterations in the insulin dosage were gauged by the degree of glycosuria in urine specimens collected in four-hour fractions.* The intravenous feedings were continued until the subsidence of postoperative nausea permitted the patient to take liquids in his usual diabetic formula. This was usually permissible by the second or third day, and a soft diet was ordinarily tolerated by the third or fourth day.

Excessive insulin doses were avoided to conserve the liver glycogen supply and to prevent hypoglycemic reactions, especially in the first forty-eight hours after operation. Increases in insulin doses during this period were based more on ketonuria than on glycosuria and hyperglycemia. Attack on the glycosuria and hyperglycemia was made when it appeared certain that the patient could tolerate his usual diet. Most of the patients were maintained on "regular" (unmodified) insulin. Two patients (F. Ha. and E. S.) who had been controlled on protamine zinc insulin with supplemental doses of regular insulin as required by ketonuria and glycosuria prior to operation were continued on this combination.

RESULTS

Twenty-three operations were performed on eleven patients without any immediate deaths. Three patients received three-stage thoracoplasty; six had two-stage operations; and two had one-stage operation. Six patients had sputum positive for tubercle bacilli before operation. Four became negative after surgery. In one exception (A. P.) the source of positive sputum after operation was found to be ulcerative tuberculous bronchitis. The sputum in this patient was converted after cauterization of the bronchial ulcers with 30 per cent silver nitrate solution. The other patient (E. S.) who continued to have positive sputum after her first stage has refused further surgery. Of the five patients who were negative before operation, two presented single instances each of positive sputum after operation. One (N. M.) had a positive sputum one week after the second stage and has been negative since. The other (D. P.) had a single positive sputum by culture six weeks after the second stage but was subsequently negative.

Three patients are working; five are convalescing in the hospital; and three are dead. The earliest postoperative death occurred ten weeks after the last stage from massive hemoptysis. The longest postoperative period of the patients now in the hospital has been thirteen months; the shortest has been two months. Of the three patients discharged, one (G. H.) had the last stage five years ago; one (A. P.), four and one-half years ago; and the other (F. Ha.), two years ago.

Changes in the Carbohydrate Tolerance.—Altnough the tuberculosis was controlled and the sputum rendered negative in most instances, the diabetic status did not show consistent appreciable improvement. In six instances the carbohydrate tolerance was lowered subsequent to surgery. It was increased in three patients and unchanged in two.

*Five units if negative, 10 units if trace to +, 15 units if ++, 25 units if ++++.

This evaluation of carbohydrate tolerance change was based on the ratio of grams of available glucose in the diet to the number of units of insulin before the first stage and after the last stage when the patient was adjudged to be in reasonably satisfactory control. Six patients required an increase in the insulin, and, of these, two required also a decrease in the carbohydrate in the diet to effect control. In three instances the insulin was decreased, the diet remaining the same. Two patients required no insulin before or after surgery except in the immediate postoperative period.

The decrease in the carbohydrate tolerance noted in this group despite the improvement in the tuberculosis is not entirely surprising. Infectious and surgical "assaults" in the diabetic often leave their mark by a lowering of the carbohydrate tolerance. This was demonstrated by John[8] in a report of several such cases, each covering a period of years. In these he showed that after infectious diseases, operations, or other disabilities, the diabetic status may become more severe. This does not preclude, however, the return of the patient to his usual activities, inasmuch as the situation can be met by appropriate dietary and insulin adjustments.

The length of time necessary to control the diabetes following each stage of thoracoplasty tended to lessen from the first to the last stage performed. This appears to us to be more than a coincidence and may be based on more than one possible cause. First, the initial stage thoracoplasty is a longer operation attended by a greater amount of tissue manipulation, surgical shock, and a longer duration of anesthesia. Second, there is a greater amount of collapse produced in the diseased lung by the first stage operation, with an accompanying autotuberculinization. This cause of post-thoracoplasty thermal reaction is discussed by Tuttle and his associates[9] and may account in part for a postoperative toxicity and temperature elevation. It was found in our review that the average *height* and *duration* of fever (over 99.6° F.) is approximately the same following the first and second stages. Hayward and Duncan[10] studied the effect of alterations in the metabolic rate on the action of insulin and found that the duration of insulin action is shortened by increasing the metabolism. These authors point out that the inhibition is not due to the actual presence of fever as much as to the presence of the toxic state. In this respect, they concur with Rabinowitch.[11] In the surgical procedure under discussion, an additional amount of collapse of the diseased lung is also obtained with the second stage operation with an added degree of autotuberculinization. The second stage operation is, however, a shorter and less immediately shocking procedure, and this may explain the shorter time required to stabilize the diabetes after this stage.

TABLE I

TABULATION OF CASES

PATIENT	SEX	AGE (YR.)	CLASS OF		DURATION OF		OTHER COLLAPSE PROCEDURES	NO. OF STAGES	PERIOD SINCE OPERATION*	INSULIN (UNITS)		AVAILABLE GLUCOSE IN DIET (GM.)		RESULTS
			TUBERCULOSIS	DIABETES	TUBERCULOSIS	DIABETES				BEFORE OPERATION	AFTER LAST STAGE	BEFORE OPERATION	AFTER LAST STAGE	
G. H.	F	27	II	Moderate	6 yr.	4 mo.	Pneumothorax and phrenic	2	5½ yr.	35	43	170	170	Living, discharged
C. L.	F	22	III	Moderate	5 yr.	7 yr.	Bilateral pneumothorax and right phrenic	3	4½ mo.	39	49	155	135	Died (heart)
A. P.	F	33	II	Severe	4 yr.	1 yr.	Pneumothorax, unsuccessful	3	5¼ yr.	60	40	205	205	Living
A. P.							After cauterization of bronchial ulcers				50		185	Living, discharged
F. H.	F	48	III	Moderate	10 yr.	5 yr.	Pneumothorax and phrenic	2	2 yr.	37	50	155	155	Died (coronary thrombosis)
M. S.	F	50	III	Moderate	1 yr.	6 mo.	Pneumothorax	2	9 mo.	38	33	180	180	Died (hemorrhage)
L. T.	F	48	III	Moderate	3 yr.	2 mo.	Pneumothorax and phrenic	3	2¾ yr.	24	0	170	170	Living and in hospital

F. Ha.	M	48	II	Severe	15 yr.	6 wk.	Pneumothorax and phrenic	2	2¼ yr.	54	60	215	190	Living, discharged
J. W.	M	48	III	Mild	10 yr.	2 yr.	Pneumothorax and phrenic	1	1 yr.	0	15	255	255	Living and in hospital
N. M.	M	39	II	Mild	2 mo.	2 yr.	Pneumothorax, unsuccessful	2	7 mo.	0	0	185	185	Living and in hospital
E. S.	F	45	II	Mild	5 yr.	1½ yr.	Phrenic	1	1 mo.	20	35	215	215	Living and in hospital
P. P.	F	49	III	Mild	6 mo.	1 yr.	Pneumothorax, unsuccessful	2	3 mo.	0	0	210	212	Living and in hospital

*From operation to Oct. 1, 1939.

TABLE II

SUMMARY

SEX		AV. AGE (YEARS)		DISEASE				AV. AVAILABLE GLUCOSE (GM.)		INSULIN (UNITS)		STATUS	
				DIABETES		TUBERCULOSIS							
Males	3	Males	45.0	Mild	4	Moderately advanced (II)	5	Before operation	192.27	Before operation	29.9	Living	8
Female	8	Females	40.25	Moderate	5	Far advanced (III)	6	After operation	188.08	After operation	31.2	Discharged	3
												In hospital	5
		Group	41.54	Severe	2							Dead	3

Third, between each stage, an interval of three or four weeks, there is some improvement in the tuberculous process with a lessened degree of toxicity from this source, reflecting itself in an improvement in the diabetes.

Example of Treatment of Diabetes.—The management of the diabetes in Case 1 (G. H.) is presented in detail.

A white female, aged 27 years, had moderately advanced tuberculosis and moderately severe diabetes mellitus. Left artificial pneumothorax failed to close cavities in upper left lobe; left phrenicectomy was performed, but this failed to close a large cavity which was held open by a pleural adhesion.

Left first stage thoracoplasty was performed, the first three ribs being removed on Dec. 7, 1933. Prior to operation the diet consisted of 70 Gm. protein, 120 Gm. carbohydrate, 120 Gm. fat; the total available glucose was 170 Gm. The fasting blood sugar was 150 mg. per cent, and the urine was sugar free with 35 units of regular insulin given in two doses. On return from the operating room, the patient was given 4 ounces of orange juice every three hours (12 Gm. of available glucose per feeding) and insulin preceding each feeding according to the degree of ketonuria and glycosuria. By the second day after operation, the patient had occasional traces of sugar in the urine and was able to tolerate a soft diet. On the ninth postoperative day, she was given four doses of insulin and was taking her usual full diet. She was sugar free after the tenth day and remained so until the time of the second stage operation. This was performed on Jan. 16, 1934 (forty days after the first stage). The fasting blood sugar on this day was 82 mg. per cent. On return from the operating room she received 250 c.c. of 10 per cent dextrose in normal saline by intravenous injection with 13 units of regular insulin given subcutaneously. Additional fluid was administered as tap water by proctoclysis. Four hours later 500 c.c. of 10 per cent dextrose in saline was given by intravenous injection with 25 units of insulin subcutaneously. This was repeated every four hours until midnight. Throughout this day the urine was heavily laden with sugar, and ketone bodies were present in faint traces to traces. On the second day after operation liquid feedings (orange juice and ginger ale) were given every three hours in 4-ounce quantities. Fractional urine specimens collected before each feeding and tested for sugar and ketone bodies determined the amount of insulin given before each feeding. By the third day after operation, a soft diet was tolerated in four feedings with four doses of insulin during the twenty-four-hour period. The urine presented only occasional traces of sugar thereafter. The diabetes was well controlled during the remainder of her stay in the hospital. She was discharged eight and one-half months after the operation. At this time the diet and available glucose were essentially unchanged, and the insulin requirement was 43 units in two doses. The weight at the time of discharge was 58.6 kg. (an increase of 3.6 kg. from the preoperative weight).

This was the first patient with diabetes to receive thoracoplasty at this hospital. It will be noted that the procedure after operation changed from the administration of frequent feedings to the administration of glucose in normal saline intravenously every four hours. The latter method is the one used in subsequent cases. We believe it is preferable to early feedings because it provides a greater amount of fluid (especially as chlorides) parenterally and because it provides certainty of retention of glucose in the event of postoperative nausea and vomiting. The data on this and the other cases are summarized in Tables I and II.

SUMMARY

Eleven cases of thoracoplasty in patients with coexistent diabetes mellitus and pulmonary tuberculosis have been reviewed. The tuberculous process was well advanced in all cases, and the diabetes was mild in four instances and either moderately severe or severe in seven.

All patients tolerated the surgical procedure well. There were no immediate deaths postoperatively. Three of the group died, however, at a later date. One death four and one-half months after the last stage was attributed to cardiac dilatation and degeneration resulting from extensive pulmonary fibrosis and reduced lung volume (necropsy performed). This patient also had pneumothorax contralateral to the operated side. One death resulted from coronary thrombosis two years after the last stage. The third death was caused by massive hemoptysis and occurred ten months after the last stage. The death rate in this group was 27.27 per cent. This is to be compared with a total fatality rate of 19.17 per cent reported by O'Brien and associates[12] in a group of nondiabetic tuberculous patients receiving thoracoplasty. The tuberculous process was checked in seven instances. In six patients the diabetes became more severe, as determined by increased insulin requirements and numerically expressed by a decrease in the ratio of available glucose tolerated to insulin required. Three patients had a decrease of insulin dosage, and two others required insulin for only a few days immediately after operation.

The thought is expressed that the surgical shock and attendant autotuberculinization following the first stage make the control of the diabetes after this stage more difficult, and the lesser shock of subsequent stages is reflected in greater ease of control. A tendency to bleed excessively was noted at operation and may be due to the arteriosclerosis commonly present with diabetes.

As in the nondiabetic tuberculous patient, thoracoplasty is a useful therapeutic procedure for the control of the tuberculous process, but every effort for satisfactory control of the diabetes prior to each stage of thoracoplasty should be exerted. Careful preparation and close postoperative observation of the diabetic status are important to insure favorable results.

CONCLUSIONS

1. The patient with coexistent diabetes mellitus and pulmonary tuberculosis commonly tolerates extensive chest surgery satisfactorily.

2. In spite of this observation, it is evident that the increased control of the infectious process does not necessarily reflect itself in an improved diabetic status.

3. Careful observation of the diabetes mellitus coupled with understanding surgery remains of paramount importance in the treatment of the group discussed.

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EARLY HISTORY OF EXTRAPLEURAL THORACOPLASTY FOR PULMONARY TUBERCULOSIS IN NEW YORK CITY

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IN THE early days of thoracoplasty it was difficult to obtain permission to have patients with pulmonary tuberculosis admitted into general hospitals for surgical therapy. This was well illustrated by the experience of the founder of the American Association for Thoracic Surgery, the late Dr. Willy Meyer, at the Lenox Hill Hospital, New York. It would seem of interest to record here some of those early difficulties.

In the fall of 1918, Dr. Edward Baldwin and Dr. Charles Trembley referred to Dr. Willy Meyer a young man, L. S., who had been at the Trudeau Sanatorium in Saranac Lake for advanced tuberculosis of the right lung and who was considered a hopeless medical case. Thoracoplasty was considered as a last resort, and he was referred to the Lenox Hill Hospital for operation. Hospitals to which such cases were referred from tuberculosis sanatoriums were general hospitals without a special chest or tuberculosis division.

Dr. Willy Meyer had vainly attempted since 1911 to get permission to admit pulmonary tuberculosis patients to the wards of the hospital. The health authorities did not permit admission of patients with active tubercle bacilli to the wards of a general hospital. The Board of Trustees, therefore, did not permit admission of active tuberculosis patients even to the private pavilion. Dr. Meyer had gained experience in these types of operations, having performed seven extrapleural thoracoplasties for advanced bronchiectasis in the years 1909 to 1918, resecting from the tenth to the first ribs in one sitting.

In 1918 he again took up the question with the Board of Trustees concerning admission of tuberculosis patients. They finally consented and gave him the use of a small, isolated room on the top floor of the hospital.

To this room L. S. was admitted in November, 1918. He had had tuberculosis of the left lung for seven years and had been in sanatoriums in Saranac Lake, Colorado, New Mexico, New York, and again in Saranac Lake. He had tubercle bacilli in his sputum from 1914 on and during 1916 and 1917 had had repeated hemoptyses. Dr. Baldwin and Dr. Trembley finally referred the patient for extrapleural thoracoplasty, since repeated attempts to produce a pneumothorax had been unsuccessful.

The patient was very delicate and dyspneic with rapid pulse, and every few weeks his temperature would rise to 103° F. and then slowly decline. Clinically, he had a far reaching tuberculous infiltration of the entire left lung with multiple cavities (Fig. 1). He had a marked cough and positive sputum.

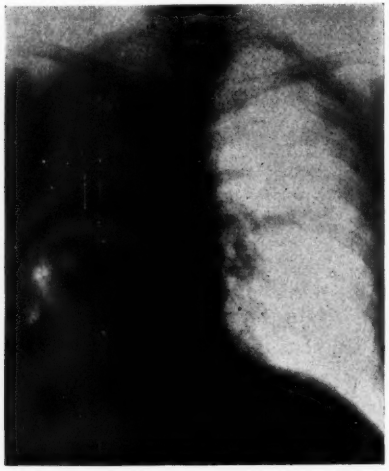

Fig. 1.—Roentgenogram of L. S. before thoracoplasty showing diffuse tuberculous infiltration of the left lung with multiple cavitation.

The first stage of an extrapleural thoracoplasty was performed on Nov. 18, 1918, under regional and local novocaine anesthesia. Ten to fifteen centimeters of the tenth to sixth ribs were resected. He made an uneventful recovery.

Seventeen days later, on Dec. 5, 1918, the second stage was performed. The fifth to second ribs were resected. Recovery once more was uneventful. He was out of bed on the sixteenth postoperative day. Fever, cough, and sputum had rapidly diminished. He gained weight and was referred back to Saranac Lake for further medical treatment. From Saranac Lake in May, 1919, this patient (Fig. 2) wrote: "I have met a number of patients in town where the doctors have advised a similar operation to mine, but they lack the courage to have it done. It seems a pity that they will not take the opportunity when physically fit, but prefer to wait and have it done as a last resort when it may be too late. It is amusing to note that as I walk along these streets I am pointed out as the ribless wonder." It is fascinating that this was the case only as short a time ago as 1919.*

*Some of the older members of the American Association of Thoracic Surgery will recall this case, as the patient was presented by Dr. Willy Meyer at the second annual meeting of the association held in Atlantic City in 1919.

L. S. regained his full health. Some time later he moved to Arizona where he is now living. I saw him during the summer of 1940, and his sputum was negative and practically absent. He is fully rehabilitated, is happily married, and has two children. In 1938, he went to the Mayo Clinic, where an x-ray photograph of his chest was made. Through the courtesy of Dr. Stuart Harrington, the accompanying roentgenograms were sent to me (Figs. 3 and 4).

Fig. 2.—L. S. in 1919, five months after thoracoplasty.

That was the beginning of extrapleural thoracoplasty for tuberculosis at the Lenox Hill Hospital, where now many thoracoplasties are performed. In concluding his paper in 1919, Dr. Meyer said, "Thus hope, well-founded hope, may still be held out to a certain number of these patients who have been battling bravely for years and formerly could see no way out."

From 1919 on Dr. Meyer attempted to have the ruling of the New York State Board of Charities pertaining to the admission of tubercu-

losis patients to the wards of general hospitals changed, but in 1925 the ruling still forbade it. In 1924 he wrote, "It is to be hoped that many of the able surgeons of this country will take up this work. It certainly would be deplorable and a hardship for these patients and their relatives if they were compelled to make long journeys in order to reach a surgeon who has training in this work."

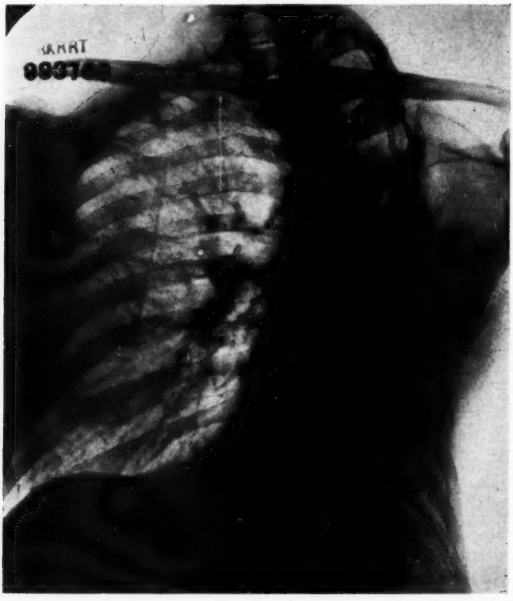

Fig. 3.—Roentgenogram of L. S. taken at the Mayo Clinic in 1937. Note that the first rib was not originally resected. In spite of this there was satisfactory collapse of the lung.

Convinced that advanced tuberculosis of the lungs had become a borderline disease, Dr. Willy Meyer continued his efforts to have the New York State Board of Charities ruling changed, hoping thereby that the several other states which had a similar faulty ruling would follow suit. The years in which these rulings were made were in the early years of the public health crusade against tuberculosis. In those years in the battle to prevent the spread of the disease no one thought of the possible use of surgery in the treatment of pulmonary tuberculosis.

Dr. Meyer felt that, in the light of the further evolution, these rulings were inadequate and unjust, as greater susceptibility in contact tuberculosis evidently exists in the young than in the mature person.

Patients for operation were scant in those years in spite of the thousands that Dr. Meyer felt were in dire need of operative help. Once the fundamental principle had been grasped, he felt that there would be many cases for operation.

Having encountered fourteen years of continued and obstinate obstruction to his efforts to have the New York State Board of Charities ruling changed, he decided upon a more active campaign to gain his ends, especially following the inspiring symposium on the subject at the Thoracic Association meeting held in Rochester, Minnesota. He therefore addressed the National Tuberculosis Association to enlist

Fig. 4.—L. S. in 1937.

their cooperation in having the ruling changed. The objectionable ruling read: "*Rule 20.* No patient shall be retained at public expense in any hospital wholly or partly under private control unless provision is made for such patients as follows: *C.* In the case of patients afflicted with tuberculosis, in wards completely segregated from any and all wards occupied by patients not having the disease." In 1923 this was modified to read: "In the case of patients admitted for the treat-

ment of tuberculosis, in rooms or wards completely segregated from any and all wards occupied by patients not having the disease."

On Jan. 21, 1925, Dr. H. A. Pattison, the supervisor of the medical service of the National Tuberculosis Association, received a letter from the New York State Board of Charities in which they stated that they did not deem it desirable to modify the ruling concerning the care of tuberculosis cases.

In assisting Dr. Meyer in the campaign, the late Dr. S. Adolphus Knopf had found that thirty-eight of forty-eight states replied to his circular letter and only four of the thirty-eight had a ruling similar to that of New York. Hereupon, the New York State Board of Charities left the entire matter open, and on March 23, 1925, Dr. Pattison was notified that the New York State Board of Charities had appointed a special committee to confer with those gentlemen most interested. Dr. Willy Meyer was invited to attend this meeting.

On April 21, 1925, Dr. Pattison advised Dr. Meyer that the rule had been amended as follows: "*C.* In the case of patients admitted for the treatment of pulmonary tuberculosis in rooms or wards completely separated from any and all wards occupied by patients not having that disease. This shall not apply to adult patients admitted for the surgical treatment of pulmonary tuberculosis. Such cases, if cared for in wards, shall be separated from other patients by a cubicle or screen."

Thus, the long battle had been won, led on by patience and perseverance. Dr. Meyer by this time was 66 years of age and, even though still a pioneer in chest surgery at this time of life, he had reached the retiring age limit for active ward service. These types of cases were therefore no longer available for him, but he rejoiced in the thought that he had won the battle for younger surgeons who he knew would carry on, as they have done.

EXTRAPLEURAL PNEUMOTHORAX

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FOR the past year, when performing an extrapleural pneumonolysis, we have used a novel approach which has proved to be so much easier than the methods previously described that it seems worthy of record.

In addition, the location of the best plane of cleavage between the parietal pleura and the chest wall was most puzzling when we first undertook this operation in 1937. When we consulted the literature, we were led into difficulties by descriptions of the endothoracic fascia as a definite fascial layer which must be penetrated in order to gain the proper plane for stripping. Consequently an investigation was carried out at autopsies on cases of pulmonary tuberculosis. The exact relation of the parietal pleura to the chest wall was determined, and microscopic sections through these tissues were obtained. An example of such a section is illustrated by microphotographs in Figs. 3 and 4. In obtaining this section the optimum plane of cleavage between the parietal pleura and the chest wall was found, and the pleura stripped away to about the middle of the rib. The block of tissue was then taken with this point as its center. It included the rib with its periosteum, the intercostal muscles, the endothoracic fascia, and the partially detached parietal pleura. Their relationships to one another are clearly shown in the microphotographs (Figs. 3 and 4). The application of these anatomic findings to the operation is described below.

TECHNIQUE

The patient is firmly supported on the operation table with the affected side upward. An assistant pulls steadily on the arm so as to retract the scapula upward and outward as far as possible.

An incision is made along the line of the fourth or fifth rib posteriorly for a distance of about 6 inches (Fig. 1*A*).

The triangle of auscultation is located, bounded by the trapezius and rhomboideus major muscles above and the latissimus dorsi muscle below (Fig. 1*B*). The lower border of the rhomboideus major may at first seem difficult to define but, if the latissimus dorsi is retracted well downward, it will be found easily. The fascia occupying the triangle is incised, exposing the fourth or fifth rib, depending upon how far the scapula can be retracted. It is usually necessary to undercut the skin and muscles slightly to increase the exposure. The amount of bleeding during this procedure is slight and can be controlled quickly by a few ligatures, leaving a clean, dry field.

From the Muskoka Hospital for the treatment of tuberculosis, Gravenhurst, Canada.
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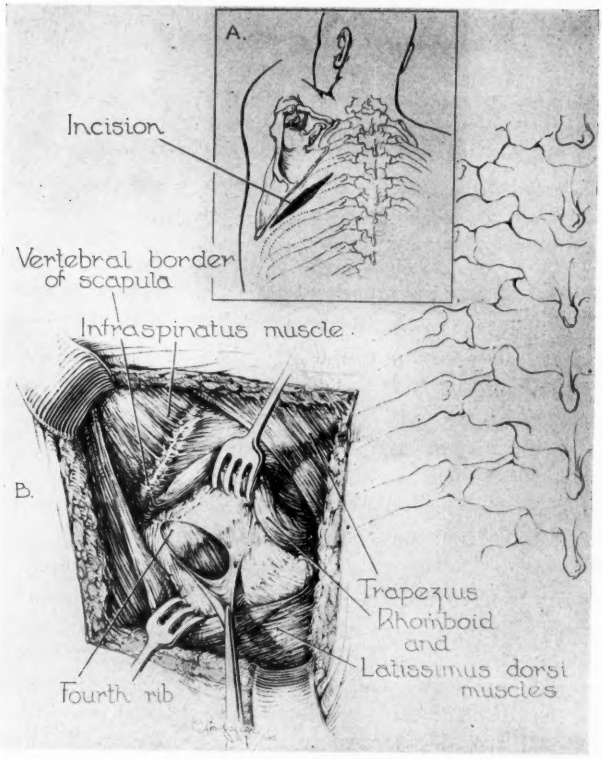
A.
Incision
Vertebral border of scapula
Infraspinatus muscle
B.
Trapezius
Rhomboid and
Latissimus dorsi muscles
Fourth rib

Fig. 1.

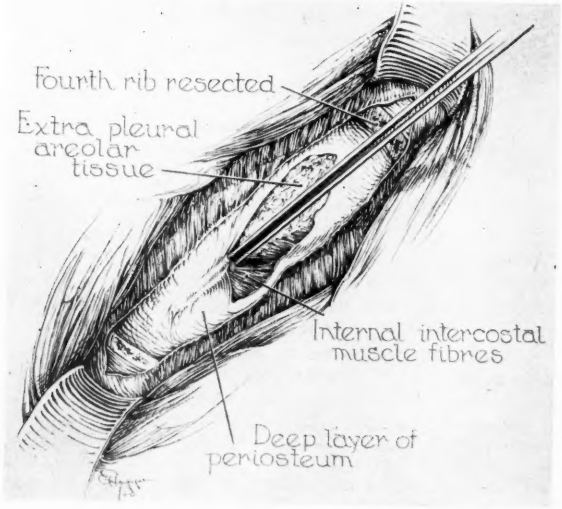
Fourth rib resected
Extra pleural areolar tissue
Internal intercostal muscle fibres
Deep layer of periosteum

Fig. 2.

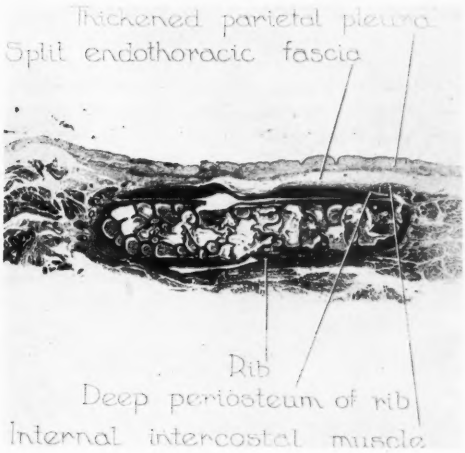

Fig. 3.—(×5).

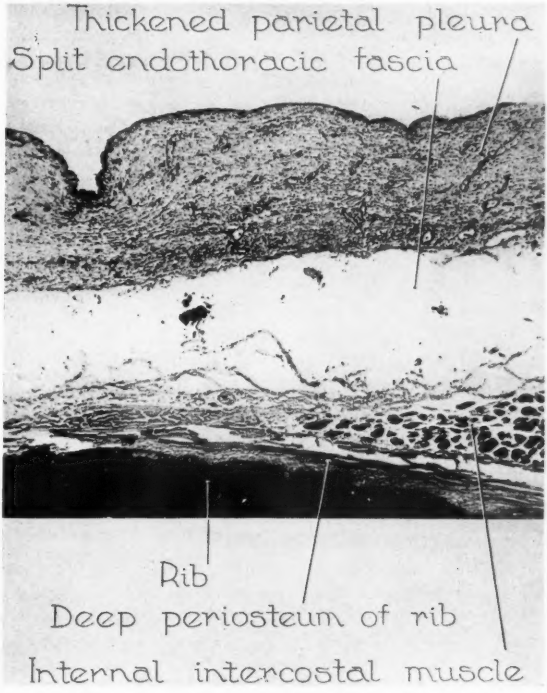

Fig. 4.—Same section as shown in Fig. 3 (×30).

The fourth or fifth rib is then resected subperiosteally from the angle outward for a distance of 10 to 12 cm. Division of the rib is not carried close to the spine since that would render closure more difficult.

After removal of the section of rib, the deep layer of periosteum is often seen to be torn (Fig. 2); if not, it is carefully incised. A few fibers of the internal intercostal muscle may be seen through this opening, but most of the space will usually be occupied by a layer of loose, fatty, areolar tissue, the so-called endothoracic fascia. Occasionally the endothoracic fascia may be so thin that the grayish-white parietal pleura is immediately visible. It is this loose endothoracic fascia (which might better be called the endothoracic areolar tissue) which is torn in separating the parietal pleura from the chest wall. This is well shown in Fig. 3. In other words, the proper plane of cleavage is immediately beneath the deep layer of the periosteum of the rib. The attachment of the internal intercostal muscle may often come well over the inner surface of the rib, and muscle fibers may be encountered after the periosteum is incised (Fig. 4). In this case the muscle fibers must be divided to expose the endothoracic fascia immediately beneath.

CONCLUSION

1. A new approach to extrapleural pneumothorax has been described which provides easy access to the field of operation and, by avoiding the division or splitting of muscles, reduces the amount of bleeding and shock.

2. The location of the plane of cleavage between the parietal pleura and the chest wall is described and illustrated with microphotographs.

LEAKAGE OF SPINAL FLUID DURING THORACOPLASTY WITH SUBSEQUENT FORMATION OF SUBARACHNOID FISTULA

A Case Report

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COMPLICATIONS following thoracoplasty, so far as the operative field is concerned, are for the most part due to either infection or hemorrhage. The profession should be familiar with the proper handling of these complications. Reporting more rarely encountered misfortunes may serve a useful purpose (1) in their prevention and (2) in their proper care when incurred.

The following case report is unique, we believe, in that no similar complication following thoracoplasty has been reported in the literature so far as we have been able to determine.

C. F. M., Caucasian, male, aged 41 years, was first admitted to Leahi Home on Sept. 6, 1934, with moderately advanced tuberculosis in the left upper lobe with cavitation and positive sputum. Following induction of artificial pneumothorax, a fairly satisfactory collapse was obtained on the left, but the apex remained adherent with the result that, although reduced in size, the apical cavity remained open and the sputum was occasionally positive. On April 5, 1935, at the patient's request, he was allowed to return to his home and to continue pneumothorax refills in the outpatient clinic.

The patient was readmitted to Leahi Home on May 30, 1938. During the interval his condition had been most satisfactory and his sputum had remained negative until the latter part of 1937 when it again became positive and remained so. Likewise, x-rays showed more definite evidence of cavitation in the left apex. He was advised to re-enter the hospital for re-expansion of pneumothorax and left thoracoplasty. Because of thickened visceral pleura, re-expansion of the lung was difficult, and by March, 1939, although the entire upper lobe was re-expanded, there was still a small hydropneumothorax pocket over the lower lobe which required occasional aspiration. It was decided at this time to proceed with thoracoplasty since x-ray films showed a definite supraclavicular cavity surrounded by dense infiltration in the left apex.

First Operation.—The first stage was done on March 22, 1939, with complete removal of the first two ribs. The operation was uneventful with the exception of a small cervical rib which was removed following removal of the first rib. The postoperative course was smooth with the exception of a small stitch abscess which delayed the second stage until May 3, 1939.

*From The Clinic.
†Director of Leahi Home.
Received for publication Feb. 1, 1941.

Second Operation.—During the second stage the accident occurred which led to the unusual complication being reported in this paper. The operation proceeded uneventfully with one exception. The third and fourth ribs were removed in the usual manner, but immediately following removal of the fourth transverse process there was a small spurt of fluid which at the time was thought to be coming from the pleural cavity directly beneath the transverse process. Since it was known that the patient had a pocket of pleural fluid in the lower part of the chest, it was believed that this fluid was intrapleural. After brief compression of this area no further flow of fluid occurred, and the incident was dismissed as being of little significance. Postoperative recovery was uneventful except that beginning the next day the patient complained of low-grade occipital headache with pain and dull aching in the posterior region of the neck. Likewise, he complained of dizziness. The blood picture was normal as were other laboratory findings. The blood pressure averaged 130/90. The temperature range was about one degree higher than before operation, ranging from 99.6° F. to 101° F. The only physical sign of importance noted was that at the angle of the scapula there was a rather large subcutaneous swelling which fluctuated. This area was not painful or tender. It was first believed to be a collection of serum. However, because of its persistence it was decided to explore this area with a needle. This was done, and about 350 c.c. of clear, thin fluid were obtained without difficulty. This fluid had the appearance of water and upon examination was found to have a specific gravity of 1.006, containing an occasional red cell and white cell but no organisms. Although immediately after aspiration the fluctuant swelling was entirely gone, in four days' time it had again recurred. Another 250 c.c. of fluid were aspirated and found by laboratory examination to be the same as previously described. It was believed then that the fluid obtained must be spinal fluid and that the patient's symptoms could be accounted for by a leakage of spinal fluid and consequent hypotension. The appearance of the fluid on the operative field at the time of biting off the fourth transverse process was recalled, and it was believed that the "leak" had taken place at this time. Conservative treatment was instituted since it was considered inadvisable to attempt surgical closure of the spinal fluid "leak" at that time. Measures instituted consisted of keeping the patient absolutely flat on his back with the foot of the bed elevated, restriction of fluids, and compression by means of rubber sponges over the region of the resected fourth transverse process. Within the next day or two the patient's symptoms of headache, dizziness, etc., ceased. Compression was released after a few days, but the foot of the bed was kept elevated for about one month. At the end of this time the patient was allowed to sit up for a short period of time and there was no recurrence of former symptoms. Bucky film of the chest at the time (July, 1939) showed that the cavity was not completely closed. It was also noted that the fourth transverse process had been clipped much shorter than ordinarily is the case. However, because of the patient's poor general condition, further surgery was postponed for the time being. He continued to make slow progress clinically. There were still low-grade fever, anorexia, extreme restlessness, and nervousness. As weeks passed, these symptoms gradually disappeared. There was no recurrence of headache, dizziness, or other signs indicating persistence of the spinal fluid "leak" until January, 1940, when the patient complained that he had had, within the past few days, two rather peculiar attacks of syncope when he had stretched his left arm upward and backward above his head in order to light his bed lamp. He experienced a feeling of faintness and lost consciousness briefly. In attempting to demonstrate this to us he extended his left arm back of his head and again became extremely faint and dizzy. Neurological examination then carried out showed his reflexes to be entirely normal and no alteration in skin sensation. A spinal puncture was done, and the fluid was found to be under 10 mm. (mercury) pressure and normal in appearance.

Queckenstedt's test raised the pressure to 18 mm. However, on raising the left arm and extending it back of the head in a position similar to that in which the patient had previously experienced faintness, the spinal pressure was elevated to 30 mm. Placing the right arm in a similar position failed to produce more than a 2 mm. rise. Although there was no swelling visible in the region of the angle of the scapula as on previous occasions, it was felt that undoubtedly there existed a sinus formation entering into the spinal canal and leading into the subscapular area and that certain positions of his arm caused scapular compression of this collection of fluid and, in consequence, the changes in spinal fluid pressure. Not having had any previous experience with such a complication, we were somewhat in doubt as to the proper method to pursue but felt that an exploration of this region should be done with an attempt at obliteration of the thecal fistula if one were found.

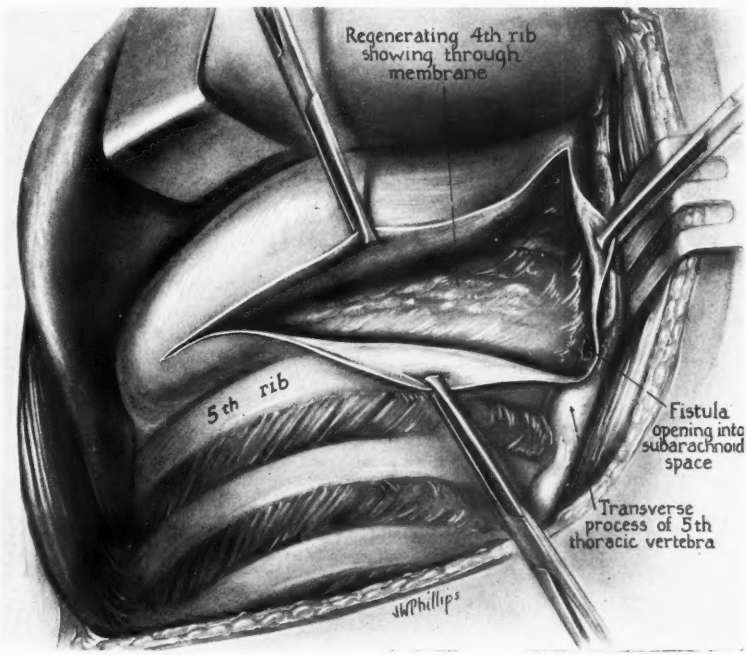
Regenerating 4th rib showing through membrane
5th rib
Fistula opening into subarachnoid space
Transverse process of 5th thoracic vertebra
J.W. Phillips

Fig. 1.—The subscapular sac which contained spinal fluid has been incised. The fistula opening may be seen.

Third Operation.—The previous incision was opened. A large pocket containing approximately 300 c.c. of clear fluid was encountered, lying beneath the scapula between the parietal pleura and intercostal muscles medially and the muscles of the chest wall laterally; anteriorly it extended into the region of the axilla and posteriorly up along the vertebral column; its extent inferiorly was limited by the fifth rib. Careful inspection of the cavity revealed a round opening approximately $\frac{2}{5}$ cm. in diameter at the site of the fourth intervertebral foramen, the point at which undoubtedly injury to the dura and arachnoid, as it extended out along the fourth intercostal nerve, had taken place at the time of the removal of the fourth transverse process. No trace could be found of any structure suggesting the intercostal nerve

in this region. Undoubtedly degeneration of this structure had taken place from injury and probably from pressure. The cavity was lined by a glistening whitish membrane which stripped away easily from the underlying structures except over the parietal pleura where it adhered to and intermingled with regenerating ribs. The lining membrane of the cavity was completely removed up to the region of the fourth intervertebral foramen. Examination of the lining membrane was later carefully made to see whether it was covered by endothelial cells, an outgrowth from the theca. The microscopic picture did not suggest this origin.

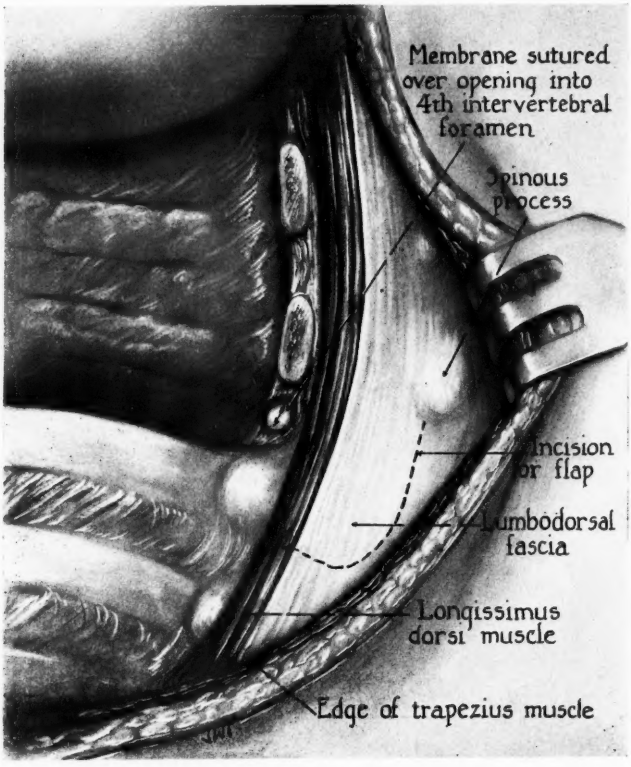

Fig. 2.—Showing steps in repair of fistula.

The fistulous opening into the subarachnoid space was dealt with by separating the membrane from the underlying bone, splitting it so it would overlap the opening, and suturing it in place with a silk suture. A flap was then fashioned from the longissimus dorsi muscle and the overlying lumbodorsal fascia, which was brought down and sutured snugly over the intervertebral foramen.

In order to further increase the pulmonary collapse in the treatment of the original lesion and to add additional support to the flap just described, portions of the fifth and sixth ribs were removed to the midaxillary line along with resection of their transverse processes. This also allowed the scapula to fall in to help in obliterating the extrapleural cavity. The incision was closed in the usual manner with two rows of continuous chromic catgut with silk for the skin and without drainage.

The postoperative course was uneventful and so far as could be determined there had not been a recurrence of spinal fluid leak. There was a suggestion of fullness in the area, but the patient experienced no symptoms as previously described during various exercises or following pressure over the operative area. However, to take care adequately of the pulmonary lesion greater collapse was deemed necessary.

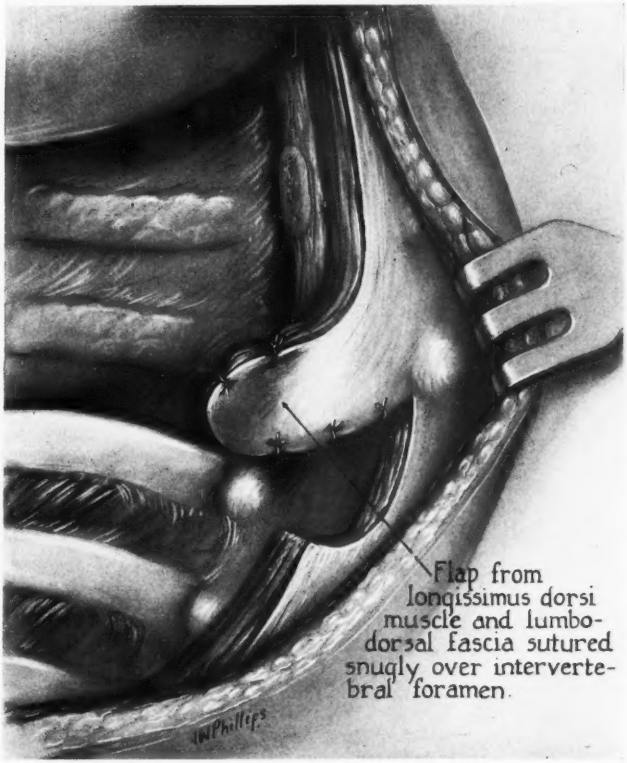

Fig. 3.—Repair completed.

Fourth Operation.—The previous incision was opened in its lower two-thirds. There was a cavity containing approximately 150 c.c. of serosanguineous fluid but no clear spinal fluid as previously. A thorough search for an opening into the spinal canal was made, but none could be seen. It was apparently healed over completely. Compression of the jugular vein did not cause spinal fluid to come into the operative field. The seventh rib was resected to the anterior axillary line along with its transverse process. Portions of the fifth and sixth ribs were also removed to the anterior axillary line. The tip end of the scapula, about 2 inches, was then resected and the muscles sewed over its tip. The incision was closed without drainage.

Since this last operation there has been no indication of recurrence of spinal fluid leak. The patient has made a steady clinical improvement. Bucky films of the chest show the cavity to be closed, and the patient's sputum has been negative for the past four months. He has been discharged from the hospital.

In retrospect we believe one can say that two conditions were present to account for the thecal fistula just described. (1) The transverse process of the fourth thoracic vertebra was not sufficiently freed of surrounding structures to prevent their injury before its removal. (2) Prolongation of the dura and arachnoid membrane along the intercostal nerve to a greater distance than normal had occurred. The process was also removed closer to the vertebra than usual.

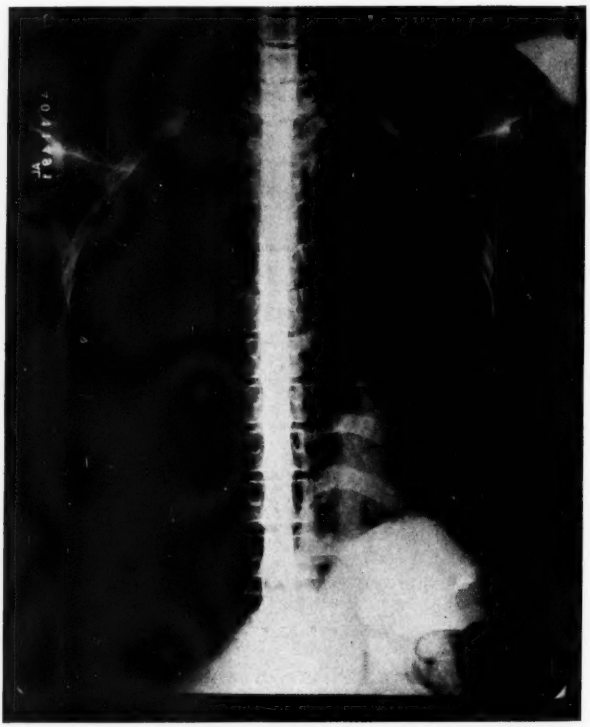

Fig. 4.—Showing probable extension of dura and subarachnoid along intercostal nerves. Three hundred cubic centimeters of 25 per cent sodium iodide were introduced under pressure into subarachnoid space by lumbar puncture.

According to Morris' *Human Anatomy,* "Spinal dura mater extends laterally and contributes to the connective tissue investments of each pair of spinal nerves and as such it passes into the intervertebral foramina and becomes continuous with the periosteum lining each." Speaking of the spinal dura: "Further, it is continuous at intervals with both the pia mater and arachnoid by way of the connective tissue sheaths of the nerve roots which are prolonged from the pia and blend with the dura mater in the passage of the nerve roots through it."

Callander's *Surgical Anatomy* says, "At the foramen magnum the outer dural layer blends with the periosteal and ligamentous lining of the vertebral canal. The inner layer is tough and fibrous and forms the dural sac investing the more delicate meninges and cord and the emerging nerve roots. It also sends out expansions over the spinal nerves emerging through the intervertebral foramina."

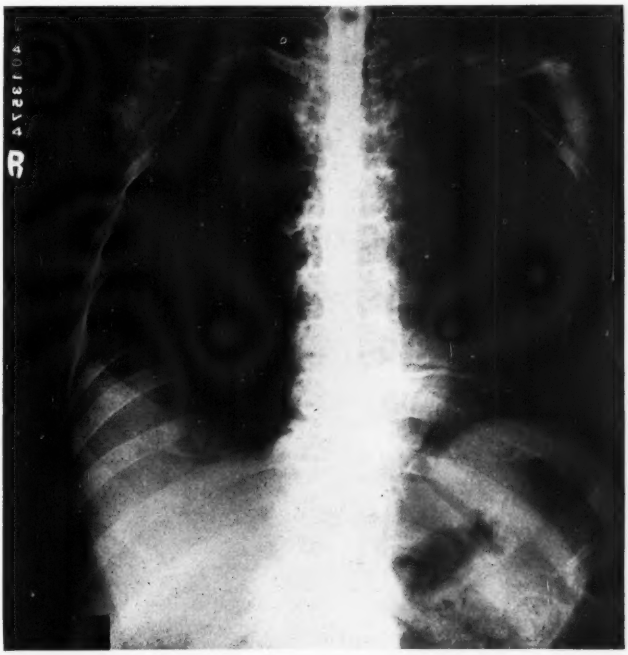

Fig. 5.—Showing extension of sodium iodide out along the eighth intercostal nerve, left. Three hundred cubic centimeters of 25 per cent sodium iodide were introduced into subarachnoid space by lumbar puncture under pressure.

In order to throw further light on the usual relationship of the subarachnoid space to the intercostal nerves, three cadavers were injected intraspinally with 25 per cent sodium iodide. It is interesting to note that in one of these cadavers the sodium iodide traveled out along the eighth left intercostal nerve for several inches. This is clearly shown in Fig. 5. Since 300 c.c. of the iodide were introduced by spinal puncture under some pressure, it may well be that spinal fluid did not normally extend to this distance. However, it is suggestive that there may be and undoubtedly is a variation in this respect since the iodide solution did not extend along the other intercostal nerves farther than the intervertebral foramen and certainly not far enough to permit injury in the usual type of transverse process removal.

In conclusion we should like to emphasize the fact that injury to the subarachnoid space with resulting thecal fistula is a possibility following resection of the transverse processes in thoracoplasty if sufficient care is not exercised in freeing the process of all surrounding structures. Dural and subarachnoid extension along the intercostal nerves usually terminates at the intervertebral foramen, but occasionally it undoubtedly extends further. A successful method of repair of such a fistula is described.

HELIUM IN ANESTHESIA AND THERAPEUTICS, PARTICULARLY IN THORACIC SURGERY

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MURPHYS, CALIF.

MORE and more hospitals are being equipped with facilities for the use of helium gas to supplement inhalation anesthesia. Considerable time elapsed following identification of helium gas in the sun's spectrum, until it was found in the natural gases of the mid-western oil fields in obtainable quantities, sufficient to make its uses commercially and therapeutically economical and practical.

Barach[1] was one of the first to point the way to its therapeutic possibilities. He reported on its uses in inhalation therapy, particularly in asthma and cardiac respiratory disease, mentioned the respiratory effort taking place with congestive heart failure and edema, and showed in his work that the patient has to maintain a high expiratory pressure for a longer time when he is expelling air than when the lighter helium mixture is employed. He also mentioned the respiratory ease following oxygen concentration in helium, with the relief of dyspnea and with positive pressure as a possibility to control a non-arterial hemorrhage. In one of his earliest experiments, Barach[2] reported that plant-life growth is able to take place in an oxygen-helium mixture, with a much lower concentration of oxygen than when oxygen is used in air (oxygen being about three times more readily diffusible in helium than in air).

Eversole[3] also pointed out uses of helium in anesthesia, while Sayers and Yant[4] reported on its use, especially in industry, because of the possibility of rapid decompression following helium-oxygen mixtures used by employees working under pressures, such as those engaged in deep-sea diving, and caisson workers. Sykes and Lawrence[5] carried out experiments on themselves, using artificial obstructions, in reporting that helium was practically twice as easy to breathe as ordinary air and its effect immediately apparent. They suggested that it might reduce the amount of respiratory work done by a patient and, when administered through a rebreather apparatus, its low solubility might prevent postoperative collapse of the lung. It has also been found useful in relief of symptoms which would not respond to other medication.

In a recent article by Barach, Martin, and Eckman[6], a number of case reports were given showing patients who were treated with 50

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per cent helium and 50 per cent oxygen under pressure of 5 to 7 cm. of water and relieved of their pulmonary edema. Experimental work also shows that changes in the heart rate and arrhythmias are due chiefly to vagal tone. One of the factors influencing vagal tone is a decrease by inflation of the lung and an increase by overinflation. Our attention has recently been called to the fact that this brings about a more rapid resolution in cases of unresolved pneumonia. Just recently it has been found that the ear trouble which is the "most common subjective complaint of both airplane pilots and passengers" can be relieved or prevented by inhalations of helium and oxygen. Therefore, we see that the efficacy of helium lies in its peculiar properties, the fact that it is an inert, colorless, odorless, noninflammable, nonexplosive gas of low specific gravity, easily displacing nitrogen (molecular weights of helium and nitrogen are 4 and 28, respectively) and diffusing with great velocity through small orifices such as the smallest air passages, thus speeding up aeration of the lung because oxygen and helium have three times the mobility of air, this point having been aptly illustrated by the following simile: "The stream of air in the bronchi, compared to a stream of vehicles in a crowded highway, and that nitrogen-oxygen molecules be represented by 28 (molecular weight, nitrogen) and 32 (molecular weight, oxygen) ton trucks, while helium is represented by 4 ton trucks, and, when 28 ton trucks (that is 80 per cent of air) are removed and replaced by faster moving lighter helium (4 ton trucks), traffic as a whole is speeded up."*

In institutions equipped with gas anesthesia apparatus, machines which do not have gauges calibrated for its use, helium may be used on the nitrous oxide gauge and the reading multiplied by the factor 3.3, to determine the amount of helium delivered; this will give an accurate reading when the flow is only 1 liter or more. With flows of less than 1 liter, the rate is probably little, if any, greater than that of nitrous oxide.

The future employment of helium gas used in therapeutics depends upon the etiology and pathologic physiology of respiratory and cardiac disorders. Personally, I have been particularly interested in the use of helium in the field of thoracic surgery, especially that which has to do with the tuberculous patient, and where we have to deal with patients of low vital capacity. From an economic standpoint, closed carbon dioxide absorption is the only one adapted to the use of helium, which, being inert, serves as a diluent element, displacing other gases. Cyclopropane is potent enough to allow of its use in large enough quantities of oxygen with helium as a diluent, without danger of producing anoxemia.

It has been the custom of the anesthetist to initiate the anesthetic by first filling the bag with about 75 per cent helium and 25 per cent oxygen,

*Dr. Boothby, Mayo Clinic.

immediately placing the mask on the patient's face, and allowing him to breathe the mixture for five or ten minutes. The flow of helium is discontinued, and oxygen is increased with the addition of cyclopropane at the rate of 300 to 500 c.c. per minute; the rate which is continued is just sufficient to compensate for any leakage. A careful watch is maintained at all times to detect the slightest degree of anoxemia. It is also customary to use helium at the close of the anesthetic period by discontinuing cyclopropane and adding helium under slight pressure to aerate and clear the lungs. It is also available during anesthesia for the same purposes. This procedure also clears the anesthetic equipment of explosive gases at the end of the anesthetic period and thus reduces the possibility of accidents.

We secure our helium through one of the commercial houses in a "B" cylinder, which contains approximately 112 liters, at a cost of $1.75 per cylinder; we use about 4 liters to the case. This varies, of course, according to the length of time it is used in the individual case. Our average cost varies from ten to forty cents per case.

It is my opinion, that helium has a distinct value at times, particularly in complications which may be met in thoracic surgery (such as those mentioned by Dolley and Jones[7]). This is true especially in instances of increased demands which result from the loss of supporting ribs, decreasing the already usually low vital capacity. Helium will enhance the respiratory efficiency and tend to overcome the demands for extra oxygen because of the difference in its specific gravity. Helium also has a place in postoperative dyspnea incident to increased toxicity from compression of the diseased lung following thoracic demands for additional oxygen, since oxygen is more speedily diffusible in helium than in nitrogen or air. At times of anoxemia due to spontaneous collapse, massive atelectasis, spasmodic contraction of bronchioles occurring during bronchoscopy, and as an aid in lessening respiratory effort at times of obstruction, we find that helium has an important place in postoperative care.

SUMMARY

1. Sufficient evidence exists to show that helium is useful to supplement our armamentarium therapeutically, in the treatment of a number of respiratory and cardiac conditions, such as asthma, edema, and air sickness.

2. It has been given a place as a means to decompress divers and caisson workers.

3. It is believed to have a place in surgical anesthesia as a supplement to other gases and as a means of clearing the lungs and equipment so as to assist in avoiding atelectasis and anesthetic accidents.

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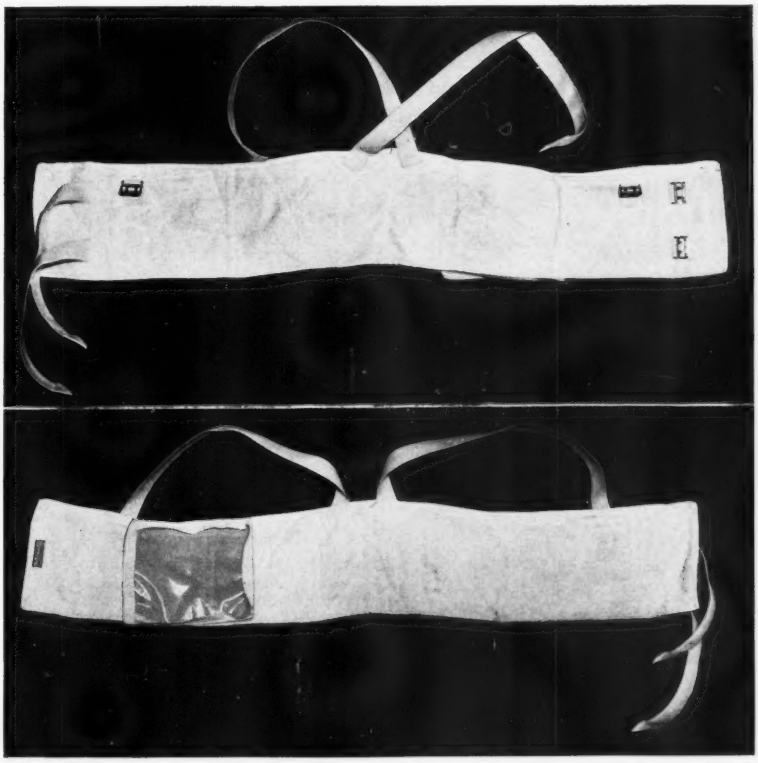
UNIVERSAL DRESSING JACKET FOR CHRONIC CHEST SINUSES

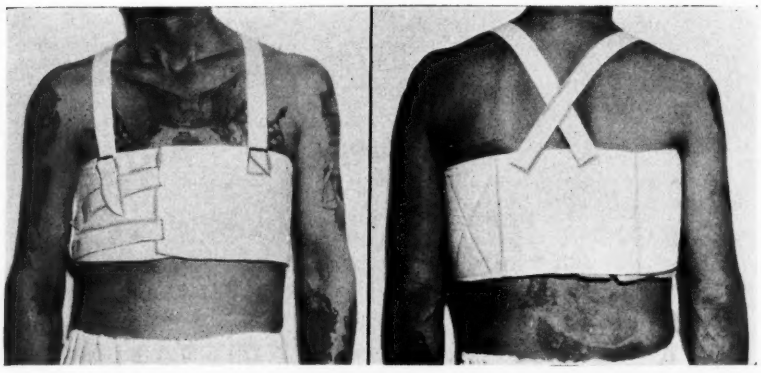
A. Lincoln Brown, M.D.
San Francisco, Calif.

THE jacket illustrated herewith has been found to be efficient and comfortable. We have used it on those patients who for one reason



or another have chronic or permanently draining chest sinuses. It was originally designed for use in sinuses following the "Eloesser flap" type of procedure.

From the Department of Surgery, Division of Thoracic Surgery, University of California Medical School.
Received for publication July 15, 1940.



The pocket, lined with oiled silk, is readily adjustable to the site of the sinus. Strips of gauze are cut from a roll and placed in the pocket as necessary by the patient. The jacket is light and obviates the continual use of adhesive tape.

The American Association for Thoracic Surgery

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Report of the Twenty-Fourth Annual Meeting of the American Association for Thoracic Surgery at Toronto

June 9, 10, and 11, 1941

Dr. Edward D. Churchill, Boston, was elected President, and Dr. Frank S. Dolley, Los Angeles, was elected Vice-President. The other officers were re-elected. Dr. Alton Ochsner, New Orleans, was elected to the Council for the four-year term, and Dr. Fraser B. Gurd, Montreal, for the one-year term.

Honorary Membership was conferred upon Dr. Norman Shenstone, Toronto, in recognition of his outstanding contributions to thoracic surgery.

Resignations were accepted from Dr. Eugene H. Pool, Dr. Paul H. Ringer, and Dr. Allen O. Whipple.

At their own request, the following Active Members were retired to Senior membership:

Dr. Elliott C. Cutler
Dr. Adrian V. S. Lambert
Dr. Hugh Trout
Dr. Chester M. Van Allen

The following Associate Members were advanced to Active membership in recognition of their work:

Dr. Thomas B. Aycock
Dr. Clarence I. Bird
Dr. Wilbur E. Burnett
Dr. James H. Forsee
Dr. John S. Harter
Dr. Ernest C. Janes
Dr. Herbert Meltzer
Dr. Lawrence W. Nehil
Dr. George F. Skinner

The following men were elected to Associate membership:

Dr. Lyman Augustus Brewer, Los Angeles, Calif.
Dr. Richard Joseph Chodoff, Philadelphia, Pa.
Dr. Frank Philip Coleman, Columbia, S. C.
Dr. Paul Geary, Plainfield, N. J.
Dr. Harlon W. Harrison, Shreveport, La.
Dr. Paul H. Holinger, Chicago, Ill.
Dr. John Randolph Paine, Minneapolis, Minn.
Dr. Richard Harwood Sweet, Boston, Mass.
Dr. William Law Watson, New York, N. Y.
Dr. Robert H. Wylie, New York, N. Y.

The Rose Lampert Graff Prize was awarded to Dr. Carl A. Moyer, University Hospital, Ann Arbor, Michigan, for his paper entitled: "Major Changes in the Fundamental Relationships of the Respiratory Drive Mechanisms During Evipal and Pentothal Anesthesia, With Special Consideration of Possible Applications to Transpleural Surgery." It was announced that the prize would be given again in 1942; the paper must be of such length to permit of its publication in the JOURNAL OF THORACIC SURGERY, and must be submitted to the Committee before Feb. 1, 1942. This year a separate prize committee will be appointed, whose membership will be announced at a later date. All papers should be submitted to the Secretary; they should not bear the name of the author, but be accompanied by an envelope enclosing his name.

The Twenty-Fifth Annual Meeting of the Association will be held in St. Louis in April or May, 1942.

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